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VOL. X. No. 4. OCTOBER, 1948

BRITISH HEART JOURNAL

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AUSCULTATION OF THE HEART*

BY

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Received July 21, 1948

Advances in our knowledge of heart disease in recent decades have generally been the result of the introduction of new techniques, either diagnostic or therapeutic. The discovery of the string galvanometer by Einthoven and the pioneering work of Sir Thomas Lewis in the field of electrocardiography clarified many problems of cardiac physiology and helped particularly to elucidate the various disturbances in the mechanism of the heart beat. Later the work of F. N. Wilson enabled us to resolve many aspects of heart muscle injury and added greatly to the diagnostic accuracy of myocardial infarction and other types of cardiac pathology. The increased use of the X-ray obviously facilitated the study of heart disease by portraying the actual size, movements, and configuration of the heart, its various chambers, and the neighbouring blood vessels. A further advance was made mainly by Sosman when calcified valves were visualized in living patients. Quite recently the electrokymograph has been introduced and although its application in clinical medicine is in its infancy, it promises to reveal interesting data about the normal and abnormal function of the heart. During the past several years venous-catheterization of the heart has been employed; it is already clear that certain diagnoses, especially in the field of congenital heart disease, can be made by this elaborate method, that were impossible without it.

Apart from the above, there are other important measurements that are often made in studying patients suffering from heart disease. Amongst these are the determination of the arterial and venous pressure, the velocity of blood flow, the vital capacity of the lung, the blood volume, etc. Sometimes one or another of these various tests proves valuable and even decisive in guiding the physician to the correct diagnosis. There are times when the serological test for syphilis or the determination of the basal metabolism prove to be the main clue in establishing a sound diagnosis and in directing effective treatment. Many of these examinations are time-consuming and expensive, but they are often indispensable. Diagnoses can now be made by the use of one or more of these procedures that were completely beyond the scope of our most learned predecessors. Nor has this advance been entirely theoretical or academic. There are numerous instances in which modern methods of study have enabled us to recognize and even to cure conditions that are amenable to effective treatment which were formerly either unrecognized or incurable. It is the function of the wise physician to choose from amongst all these procedures the one or more, preferably the simplest, that are essential in guiding his treatment.

During this period of rapid advance in technology and in therapeutics as it pertains to heart disease, one method of examination has suffered—auscultation of the heart. The interest and energy expended in these other fields, although extremely profitable, have detracted from the attention given to the use of the stethoscope. In addition, there was a more legitimate reason for attaching less importance to auscultatory findings. During the period before the first World War, physicians attributed too great importance to certain cardiac irregularities and murmurs. Largely as a result of the teaching of Sir James MacKenzie, the significance of the symptoms of heart failure (congestive or anginal) began to be appreciated. He convinced the medical profession that many cardiac murmurs and irregularities were benign and compatible with long and vigorous lives. Physicians were prone to administer digitalis and to

* The St. Cyres Lecture of the National Heart Hospital, London, July 8, 1948.

† I wish to express my great indebtedness to Dr. W. Proctor Harvey, Assistant in Medicine, Peter Bent Brigham Hospital, Boston, for his help in obtaining numerous phonocardiograms and in other ways.

restrict greatly the activities of patients, even confining them to bed, because of the detection of abnormalities that we now know were entirely harmless. The change in viewpoint that took place some thirty or forty years ago was a great step forward.

However, all this led to discrediting the use and value of the stethoscope. I recall hearing MacKenzie say that the stethoscope should be thrown away, because it had done more harm than good. To be sure, auscultation sheds very little light on whether the heart is failing or not. The only stethoscopic finding that indicates heart muscle incompetency is a diastolic gallop or possibly an alteration of ventricular systole. But what must not be overlooked is that many patients have organic heart disease, especially valvular disease, for many years before they have heart failure. They often want to know and need to know, during these early years, whether they do or do not suffer from heart disease. In some of them auscultation is the simplest and occasionally the only method of answering this question, which is important notwithstanding the fact that during the period of normal compensation one would not advise cardiac medication or unduly restrict activities if a faint murmur of rheumatic aortic insufficiency is found. Such a patient might need prophylactic penicillin therapy for the extraction of teeth to protect him against bacterial endocarditis if aortic insufficiency were present, and would not if the heart were perfectly normal. This and other matters of considerable importance to the health of the patient depend upon the accurate diagnosis of apparently minor abnormalities that auscultation may reveal.

It is surprising that so little direct experimentation has been carried on concerning auscultatory findings. Our interpretation of the significance of cardiac murmurs has largely depended upon the correlation of physical findings in the sickroom with observations carried out post-mortem. This method has been very valuable but has definite limitations. We are unable to trace back and study the anatomical and pathological changes that were present years before the patient died, and explain the findings that were present when the heart was well compensated. Furthermore, at the autopsy table we see the atonic heart and cannot readily picture what the position of the valve leaflets might have been when the heart performed its function with the existing pressure relationships. Valves that appear normal or essentially so when observed directly, may have been incompetent when functioning in the living heart. We know that the aorta can be considerably dilated when viewed fluroscopically and yet show normal dimensions post-mortem. This has been called the hyperdynamic aorta. May not similar changes be taking place within the chambers of the heart?

Another point about which accurate data are lacking is the relationship between the size of a defect and the loudness of a murmur that may result. Under what circumstances does a loud murmur represent a slight valvular defect and when is the reverse true? It would seem logical that a very loud systolic murmur of aortic stenosis would accompany a high degree of stenosis and a similarly loud mitral systolic murmur might indicate a slight mitral insufficiency, because in each case we would expect the intensity of the turbulance to be greatest when the blood stream suddenly flows through a very narrow orifice. The situation does not appear to be the same with regard to aortic insufficiency, for here it seems that a faint diastolic murmur is present with a slight regurgitation. Some of these questions could be readily answered by direct animal experimentation.

The exact position and movements of the valves, especially the auriculo-ventricular valves, during various phases of the cardiac cycle is of considerable importance in the discussion to be taken up later. Most physicians look upon the closure of the mitral and tricuspid valves as occurring in a hinge-like fashion, like the closing of a door. If that were true, it would appear that during the very early part of ventricular systole, as the valve leaflets were being approximated, there would necessarily be some regurgitation before the valve actually closed. Yandel Henderson (1912) reported some ingenious experiments many years ago, supporting the view that closure of the A-V valves took place in a manner that would prevent this possible reflux. He believed that as a result of a " jet effect " the A-V valves were drawn close together directly after auricular systole, and with ventricular systole they finally closed by a mechanism similar to the unrolling of a carpet. In this fashion there would be no regurgitation during ventricular contraction. Furthermore, it is a matter of some importance whether the valve leaflets are driven deeper into the ventricular cavity and spread more widely apart by auricular systole or whether they attain a higher position and are drawn nearer together. It may be debatable whether the experiments of Henderson can be translated directly to events that occur in the normal human heart and whether they apply with equal significance to abnormal states. In any case, there seems to be little doubt but that auricular contraction alters the exact position of the A-V valves and will have a determining effect on the position of these valves at the very moment ventricular systole occurs.

The First Heart Sound

Although many points about the mechanism of cardiac sounds and murmurs are still vague and meagrely understood, there are some clinical correlations of auscultatory findings with practical application that are not generally appreciated or utilized. Amonst these is the significance of the intensity of the apical first heart sound. It is generally taught that the first sound has two components, valvular and muscular. Experiments performed by Dock (1933) led him to the view that the first sound is entirely valvular. Observations to be discussed here indicate that if there is a muscular component it is so faint that it is essentially inaudible on ordinary auscultation. For present purposes, therefore, it can be assumed that the apical first sound is valvular in origin.

There are many occasions on which the heart sounds are decreased in intensity. It is obvious that in patients who are dying or moribund, when the heart is extremely feeble, the heart sounds become very faint. Likewise, patients with much obesity, emphysema, or pericardial effusion may have very distant heart sounds. In fact, any condition that interposes excessive tissue between the heart and the skin will necessarily decrease the intensity of the sounds as heard with the stethoscope. In all those conditions, both first and second sounds will be diminished. Excessive amounts of air or tissue between the heart and the external thorax will not affect one sound without producing a similar effect upon the other. What is more important is that the first sound can be altered (increased or decreased) without a simultaneous change in the intensity of the second sound. It is this phenomenon that deserves particular attention, for its detection enables the physician to make certain bedside diagnoses by simple auscultation that formerly were regarded as impossible without elaborate graphic methods.

The loudness of the first sound depends upon several factors. The vigor of ventricular systole is one of these, but by no means an important one. Many patients have serious organic heart disease and even failing hearts with perfectly good heart sounds. Contrariwise, a decreased first sound is common in perfectly healthy subjects. Of greater significance is the quickness or abruptness of ventricular systole. Hyperactive or loud sounds, particularly the first sound, are often heard in hyperthyroidism (Fig. 1 and 2), anæmia (Fig. 3), after a brief exercise, during certain infections (particularly rheumatic fever), and with some emotional states. In most of these conditions, if not in all, the ventricles contract briskly. On fluoroscopic examination one can often see a "snapping or hyperactive type of beat." With this there is likely to be an increase in the velocity of blood flow. Generally this is determined by measuring the circulation time from a vein in the arm to an artery in the head. What determines the effect on the loudness of the first sound, and for that matter on the production of cardiac murmurs, is not the total speed of flow but the velocity of the blood stream within the chambers of the heart and the neighbouring great vessels. It is quite likely that this may be accelerated even when the total speed of flow as measured in the ordinary way is little, if at all, affected. One would expect that the duration of mechanical systole would be slightly shortened in these hyperactive states, and the increased intensity of the first sound would result from the abruptness of closure of the A-V valves. Many years ago Samuel Gee (1908), in discussing murmurs, stated "The loudness of the sound depends upon the swiftness of the flow: the quality of the fluid and the size of the orifice are of import only inasmuch as they exert an influence upon the swiftness of the flow." This mechanism not only affects the intensity of murmurs, but also of heart sounds.

Another condition in which the first sound is notably increased in intensity is mitral stenosis. The cause of this peculiarity has been a matter of much speculation. One naturally suspects that the anatomical changes in the valve itself may influence the loudness of the sound. However, there are other influences involved. In mitral stenosis, the papillary muscles are hypertrophied and the chordæ tendineæ are shortened. The valve leaflets are probably deeper in the ventricular cavity than normally. Furthermore, the left auricle is generally dilated or hypertrophied, and with the obstruction of the valve, the filling of the ventricle occurs more slowly and gradually. Finally, the ventricle may contract with a slightly smaller volume and more abruptly. The result of these various factors is that the valve may be at an abnormal position the moment ventricular systole occurs and, as we shall discuss shortly, this influences the intensity of the first heart sound.

A final factor that determines the loudness of the first sound, and one that I believe to be the most important, is the exact position of the A-V valves at the instant the ventricles contract. It was stated above that there is some difference of opinion whether the A-V are driven deeper and wider apart or higher and closer together by auricular systole. Although I assume the first of these two premises, the fact that auricular systole changes the position of the valves can hardly be doubted. The observations to be discussed and the arguments involved are equally applicable on the basis of either theory, for the main point is that the first sound is different

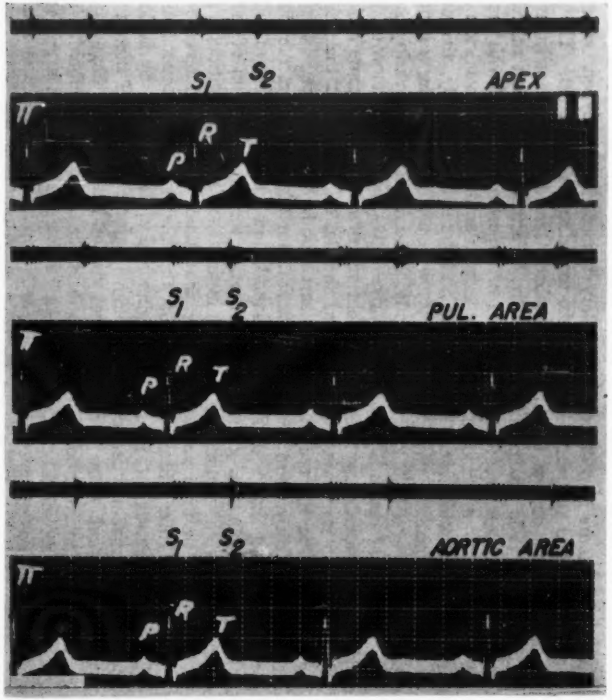

FIG. 1.—Normal heart sounds. Normal male, 40 years old. Note first sound (S–1) is louder than second sound (S–2) at apex, but the relationship is reversed at the base of the heart. This is the customary finding in normal hearts.

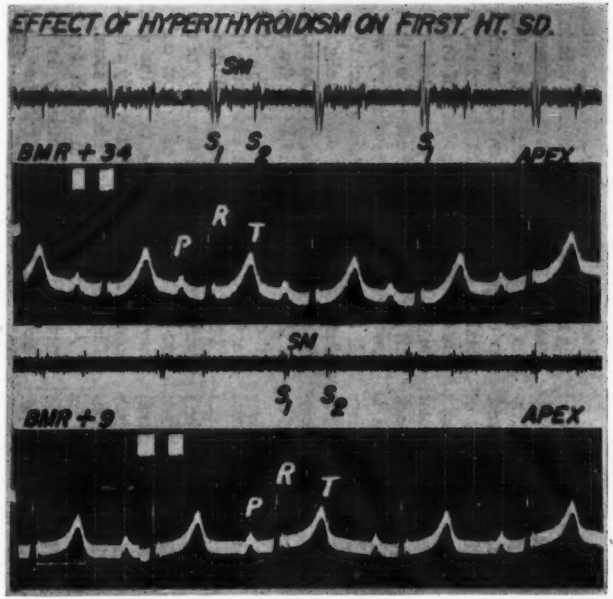

FIG. 2.—Girl, aged 18 years. Diagnosis thyrotoxicosis. Upper tracing while BMR was +34. Lower tracing about one month later, after propyl thiouracil therapy. BMR +9. Note great decrease in intensity of first sound (S–1) and also of systolic murmur (SM).

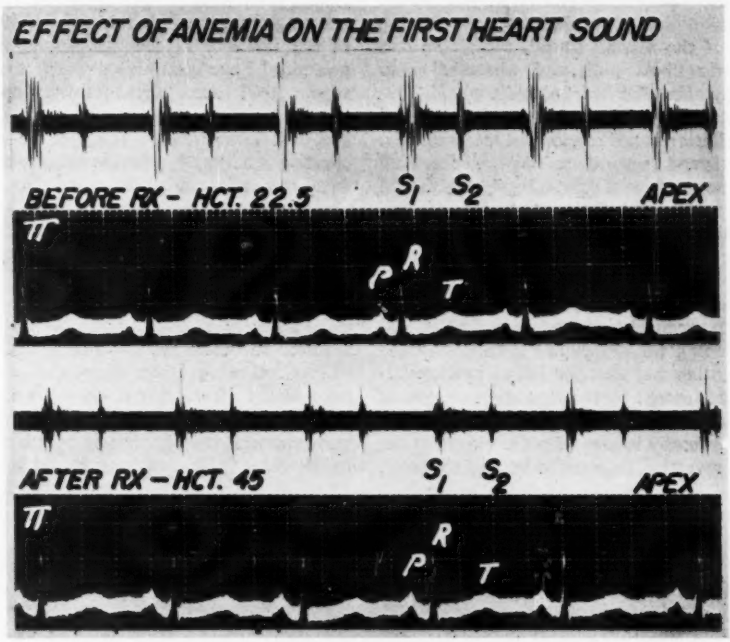

FIG. 3.—Woman, aged 68 years. Diagnosis pernicious anæmia. Upper tracing: hæmatocrit was 22·8. Lower tracing: Several months later, hæmatocrit 45·0. Note decrease in intensity of first sounds (S–1).

if the valves are in one position from what it is if they are in another. It will be assumed that the first sound is louder if the valves are deep in the ventricles and wide apart and is fainter when higher up and closer together.

If this theoretical speculation is correct, it should follow that the relation between the time of auricular and ventricular systole will have a profound effect upon the loudness of the first sound. Normally the ventricles contract 0·16 to 0·18 sec. after the auricles. Auricular systole, it may be assumed, propels the A-V valves into the ventricular cavities, and as the ventricles partially fill, the valves move upwards to a mid-position and then are abruptly closed as ventricular systole occurs. If the ventricles contract 0·08 sec. after the auricles, the valves will be caught at a lower position and the snap that results with ventricular contraction will be louder. However, if ventricular contraction occurs 0·22 sec. after auricular, the ventricles will have had a longer time to fill and the valves will be higher and more closely approximated. Closure will then result in a more feeble sound. In a word, the loudness of the first sound, according to this reasoning, ought to reflect to some degree the length of the P–R interval.

We have, therefore, a means of estimating the P–R inverval by auscultation of the heart. If conditions cited above like mitral stenosis, hyperthyroidism, anæmia, etc., which may increase the intensity of the sound, can be eliminated, an accentuation of the first sound at the apex strongly suggests that the P–R interval is shorter than normal. It has been found that the sound is loudest when the interval is about 0·04 to 0·08 sec. (Wolferth and Margolis, 1930). Contrariwise, if the sound is fainter than normal, the P–R interval is full or unduly prolonged (0·20 to 0·24 sec. or more). It does not follow that the first sound becomes increasingly weak as the P–R interval lengthens more and more. The intensity may be decreased just as much or more when the P–R interval is 0·22 sec. as when it is 0·30 sec. In estimating an increase or decrease in the loudness of the first sound it is well to compare it with the second sound. None of the above inferences can be drawn if both sounds are much decreased. One should compare the

loudness of the first sound with what one would expect to hear in that particular case, considering the intensity of the second sound, the shape and thickness of the chest wall, and whatever other conditions may affect the heart sounds.

Many auscultatory findings are often missed because auscultation is not carried out methodically. One should spend several seconds deliberately listening to one point at a time. First, one should listen to the first sound for several seconds, dismissing everything else from one's mind. Then the same procedure should be carried out for the second sound. This is continued for the interval between the first and second sound (systole) and finally for the interval between the second and first sound (diastole), listening for additional sounds or murmurs. In no other way can fine points in auscultation be detected, except by concentration on one of these four components at a time. Normally, the first sound is generally louder than the second at the apex, and the reverse is true at the base of the heart (Fig. 1).

It is surprising how accurate one can become in guessing what the P–R interval will be on the basis of the intensity of the first sound (Fig. 4). Repeatedly I have been able to predict this interval to within 0·01 sec. This information, so readily obtained by simple auscultation, has considerable practical importance. There is no other way of eliciting it except by the use of graphic methods or other complicated techniques. It would require obtaining daily electrocardiograms to detect some of the transient changes in the P–R interval that occur in myocarditis of rheumatic or diphtheritic origin. Furthermore, many hearts with a slight delay of the P–R interval may otherwise appear quite innocent, so that the general practitioner is not tempted to obtain electrocardiographic records.

The following is an illustrative experience. A man about 50 years old came to the out-patient department because he had fainted a few times. He was otherwise well, having no breathlessness or pain in the chest. He could work and walk quite well. Physical examination revealed nothing abnormal.

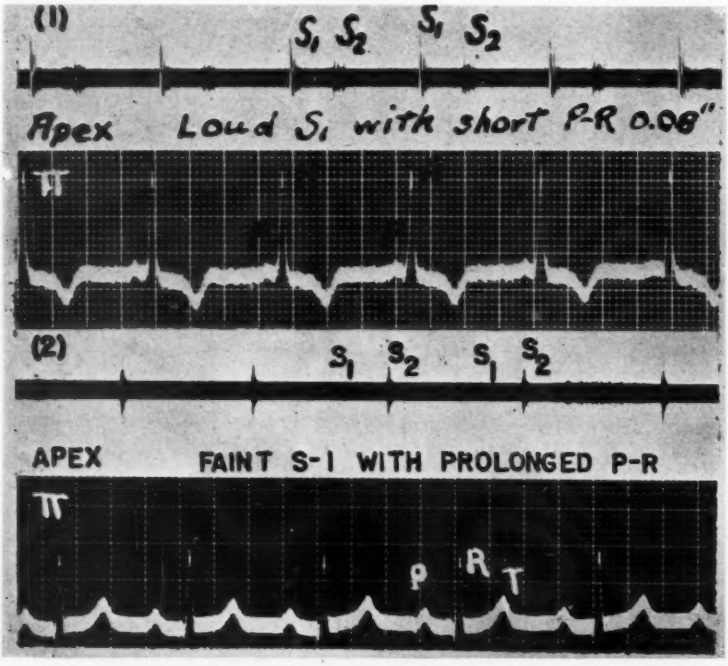

FIG. 4.—Upper tracing shows loud first sound (S–1) with short P–R interval (0·08 sec.). Patient was a man, 24 years old, with essential hypertension. Lower tracing shows almost inaudible first sound (S–1) with only slightly prolonged P–R (0·22 sec.). Patient was a 24 year old woman with Gaucher's disease.

The blood pressure was normal, the heart showed no enlargement, arrhythmias, or murmurs, and the carotid sinus was not unduly sensitive. On careful auscultation the apical first sound was extremely faint, though the second sound was normal. This important finding was entirely overlooked by the physicians who first examined him. When the same physicians were asked to re-examine the patient, paying particular attention to the first sound, they quickly realized that it was strikingly decreased in intensity. Then followed a further misinterpretation of the significance of this finding. When asked what a decreased first sound signified, the reply was that it indicated heart muscle weakness. However, this patient seemed fit and gave no evidence of myocardial incompetency.

It is obvious that many patients have grave myocardial insufficiency with normal first heart sounds and may have poor or even inaudible first sounds with perfect heart function. The old concept that a poor first sound means a weak heart muscle is entirely fallacious. In the case cited above, the interpretation was made that the P–R interval was delayed, possibly to 0·21 to 0·22 sec. An electrocardiogram was then taken directly, which showed the interval to be 0·22 sec. The practical point of this experience is that a simple auscultatory finding led me to suspect that the previous attacks of syncope were due to Adams-Stokes disease. Only later did we learn that he had previously been observed in a hospital during one of these spells, and there showed transient complete heart block.

Similarly it is possible to suspect that the P–R interval is unusually short. If the common causes for an accentuated first sound like mitral stenosis, hyperthyroidism, etc., can be eliminated from consideration, a snapping first sound is very likely to mean that the P–R interval is less than normal (Fig. 4). It may be only 0·14 sec., but more frequently 0·12 sec. or less. It has been somewhat surprising that the first sound in cases of the Wolff-Parkinson-White syndrome, though somewhat accentuated, is not as loud as one might have expected with P–R intervals as short as 0·08 to 0·10 sec. This may be due to the fact that in this condition early excitation occurs generally in the right ventricle, and that closure of the mitral valve (the more important one of the two A-V valves) takes place at a normal interval after auricular systole. In any event, there are other instances of short P–R interval that are associated with attacks of paroxysmal rapid heart action and, therefore, simple auscultation may lead the physician to recognize such cases.

There is abundant proof available that changes in the P–R interval *per se* have a profound effect on the intensity of the first sound. In an experiment in which the heart rate happened to remain unchanged after the intravenous injection of 1·0 mg. of atropine, the P–R interval was markedly decreased while the first sound became greatly accentuated (Fig. 5). In this experiment the microphone and stethocardiographic mechanism was not disturbed in any way, so that the loudness of the registered sounds could be accurately compared. A very feeble sound, which was present because the P–R interval was delayed (0·28 sec.), became quite loud fifteen minutes later when the interval shortened to 0·18 sec.

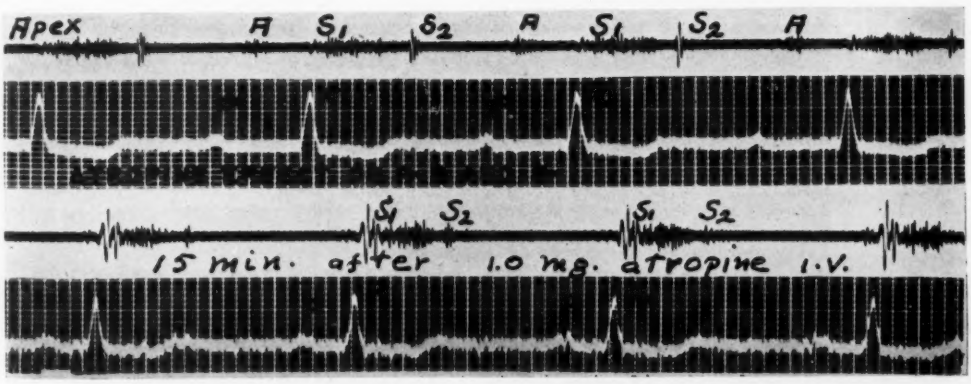

FIG. 5.—Upper tracing shows faint first sound (S–1) with long P–R interval (0·28 sec.). Lower tracing shows loud first sounds (S–1) 15 minutes after 1 mg. atropine i.v. when P–R interval was normal (0·18 sec.). Auricular sounds (A) were detected in upper tracing.

Complete A-V Block

Another and more important observation that affords proof of this general premise is the changing intensity of the first sound in cases of complete dissociation of the auricles and ventricles (complete heart block). This auscultatory phenomenon was first observed by Strazhesko (1906) and independently by Wardrop Griffith of Leeds (1912). The true explanation of the changing first sound was at first not understood. In the course of time this finding became known as the "bruit de canon" because of the explosive quality of the first sound in occasional cardiac cycles. Even when Lewis (1915 and 1925) published phonocardiograms of this mechanism (1925), he erroneously ascribed the loud sounds to the simultaneous contraction of the auricles and ventricles, inferring that a summation effect caused the accentuation. Wolferth and Margolis (1930), however, clearly demonstrated that the greatest intensity of the sound occurred when the P–R interval was between 0·04 to 0·08 sec. and not when auricular and ventricular contractions took place simultaneously.

When the ventricles are beating slowly and regularly and the auricles more rapidly, regularly, and independently, we have an opportunity of observing the effect of changing P–R relationships from cycle to cycle without any other disturbing influence. It will be found that the sounds are loudest when the interval is unusually short and fainter when long (Fig. 6). In fact, there may be occasions when the first sound is almost inaudible

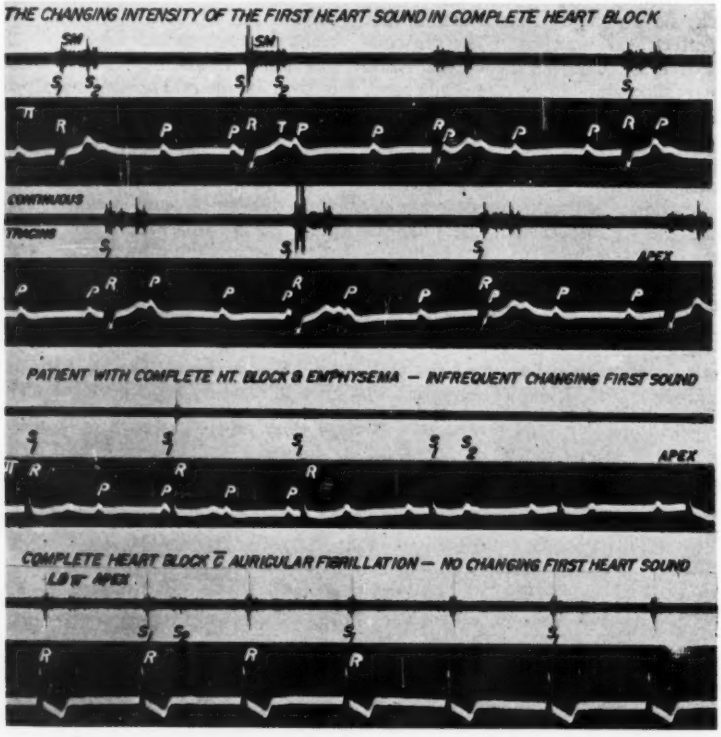
THE CHANGING INTENSITY OF THE FIRST HEART SOUND IN COMPLETE HEART BLOCK
A
CONTINUOUS TRACING
APEX
PATIENT WITH COMPLETE HT. BLOCK & EMPHYSEMA — INFREQUENT CHANGING FIRST SOUND
B
APEX
COMPLETE HEART BLOCK & AURICULAR FIBRILLATION — NO CHANGING FIRST HEART SOUND
C

Fig. 6.—(A) Upper two strips are continuous. Man, 33 years old, with Adams-Stokes disease with complete A-V dissociation. Note changing intensity of first sounds (S–1), loudest sounds coming with very short P–R intervals. Intensity of systolic murmur (SM) remains constant. (B) Second patient, aged 60, had complete heart block and emphysems. Note that although most sounds are distant, when the P–R interval is very short, S–1 is loud. (C) Lowest tracing: Man, aged 72 years. Diagnosis complete heart block with auricular fibrillation or idioventricular rhythm. Note constant intensity of first sounds (S–1).

and at other times strikingly accentuated. This changing intensity of the first sound in a slow, regular beating heart is practically pathognomonic of complete heart block. Our medical internes have been sufficiently alert in recent years to make the bedside diagnosis of complete block by means of this sign, even in cases in which the ventricular rate was not 30 to 40 but 50 to 75, as occurs occasionally in acute coronary thrombosis. The marked diminution, and at times the complete absence of the first sound in occasional cycles when the P–R interval is delayed, afford additional proof that the muscular component must be of very little importance in the causation of the first sound. These cycles are just as vigorous, and are accompanied by a peripheral pulse of the same volume as those producing a loud sound.

One would predict that if there were complete heart block and the auricles were fibrillating the first sound would maintain a constant intensity. This would necessarily follow because, although the ventricles are beating independently, the auricles are not contracting, and therefore would not alter the position of the A-V valves at the time of ventricular systole. That this is the case is well illustrated in Fig. 6C.

Paroxysmal Ventricular Tachycardia

There are other conditions in which the contractions of the auricles should produce alterations in the intensity of the first sound. In most cases of paroxysmal ventricular tachycardia, the auricular rhythm is independent of the ventricular and controlled by the normal sinus pacemaker. As would be expected, the intensity of the first sound varies in different cycles (Fig. 7). This auscultatory sign was described some years ago (Levine, 1927), and may enable the examiner to distinguish the auricular from the ventricular type of paroxysmal tachycardia. In the common variety of paroxysmal auricular tachycardia (without heart block) the heart sounds are not only rapid (150–250) and regular, but are constant in quality and intensity. In ventricular tachycardia, the rate is also rapid (150–250), generally regular (though frequently slight irregularities may be present) but the intensity of the first sound may vary considerably. In fact, obvious alteration in the first sound may be detected when the ventricular cycles are perfectly regular. This feature, together with the failure to influence the ventricular rate by vagal stimulation, helps to distinguish, on simple bedside examination, the ventricular from other types of paroxysmal rapid heart action.

Auricular Flutter

The character of the first heart sound has recently been investigated in auricular flutter. In many cases of auricular flutter before therapy is instituted, the ventricular rate is exactly one-half of the auricular. Under these circumstances every other impulse from the auricles is blocked. The rhythm of the auricles is perfectly regular though rapid (250–350), and the ventricular response is also perfectly regular (125–175). The interval between the auricular impulse that is not blocked and the subsequent ventricular contraction is constant from cycle to cycle. The result is that the first heart sound is constant in character, just as it is in paroxysmal auricular tachycardia or in normal sinus tachycardia (Fig. 8). However, there are instances of flutter in which the ventricular rate, though rapid,

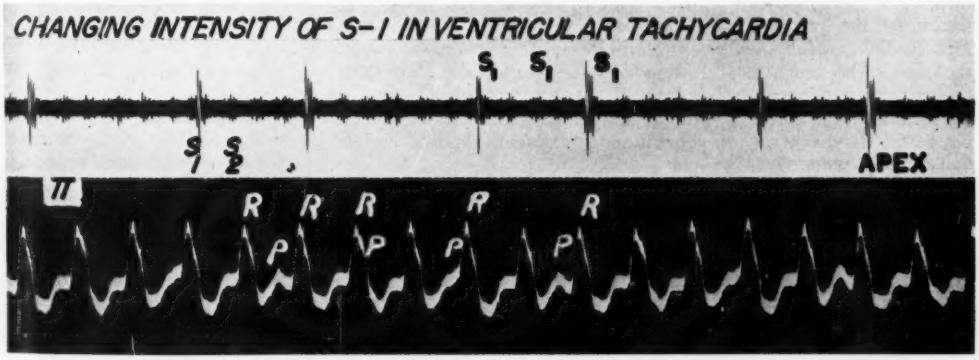

Fig. 7.—Man, 63 years old. Diagnosis paroxysmal ventricular tachycardia. Note changing intensity of first sounds (S–1) with auricles and ventricles beating independently. Note loudest first sounds come with short P–R intervals.

is not absolutely regular. Slight differences may be detected in the interval between the auricular and ventricular impulses in different cycles. This would necessitate alterations in the intensity of the first sound (Fig. 8 and 9). Such changes have been observed, and have been shown to be due to the variation in the A-V relationships and not to the length of the diastolic pause (Harvey and Levine, 1948).

A further auscultatory finding in auricular flutter is the detection of auricular contractions. Not only may these faint sounds be audible during the long diastolic pauses when the ventricles have slowed, but they may be heard during systole (Fig. 9). Occasionally they are quite loud and confusing so that the observer may be misled into considering the mechanism as due to auricular fibrillation. A well known simple bedside method of distinguishing the irregularity of flutter from that of fibrillation is the effect of exercise. When the heart accelerates after a brief effort, the irregularity of flutter is likely to change to a more rapid but regular rhythm while that of fibrillation continues to be irregular though more rapid.

A peculiarity of auricular flutter that is less well known is the response to vagal stimulation. When the heart rate as auscultated over the præcordium is perfectly regular and rapid (140–175), it is often possible to slow the ventricular rate temporarily by carotid sinus pressure or other means of vagal stimulation, such as holding a deep breath. In auricular flutter, after a brief period of slowing, the original rapid rate is often resumed in a *jerky* fashion (Fig. 9). In paroxysmal auricular tachycardia without block, vagal stimulation will have no effect whatever or completely arrest the attack. In normal sinus tachycardia, if any effect is produced, there will be temporary slowing with a *gradual* return to the previous rate, and in ventricular tachycardia carotid sinus pressure will never alter the rate. The temporary slowing and the *jerky* return of the previous tachycardia is fairly characteristic of auricular flutter, and helps the physician to diagnose this condition by simple bedside examination.

The above auscultatory findings prove very useful, not only in the original recognition of auricular flutter, but in following its progress under treatment. While administering digitalis or quinidine, it is often important to increase, decrease, or omit the drug depending upon the changes in the arrhythmia. It might be necessary to take frequent cardiograms to know whether flutter is still present or whether the rhythm has reverted to normal or to fibrillation. A day or two after administering quinidine or digitalis to a patient with flutter, for example, the heart may be found quite regular at a rate of 70. One might readily conclude that the mechanism is normal, but in point of fact flutter may still be present with an auricular rate of 280 and a pure 4:1 block. A brief exercise test is likely to clarify the diagnosis. If flutter is still present, the rate may quickly jump to 140 and remain regular for a short interval or become temporarily irregular and rapid (Fig. 9). If the original slow regular rate had been due to a normal sinus rhythm, temporary acceleration would have occurred smoothly and could only by accident have reached the rate of 140, which is an exact multiple of the original rate of 70. These simple guides may quickly enable the physician to make decisions, and will save considerable time and expense.

Pulsus Alternans

Pulsus alternans is a phenomenon detected in the peripheral arteries in which the pulse, though perfectly regular, alternates in volume. It is generally believed to indicate a fairly grave disturbance in myocardial function. It is even more easily detected on auscultation below the pressure cuff when determining the blood pressure. Here the sounds will alternate in intensity, and when the condition is very marked, only the stronger of the alternate beats will be audible at the very highest levels of pressure. What has not been currently appreciated is that the same phenomenon may be detectable on auscultation of the heart. Not infrequently the intensity of the heart sounds or of an accompanying systolic murmur may alternate from cycle to cycle (Fig. 10). When this is heard, it has the same significance and is due to the same disturbance in contractility as obtained when pulsus alternans is observed in the peripheral arteries.

Gallop Rhythm

In identifying a gallop rhythm by auscultation, it is always important to distinguish the systolic from the diastolic type. Whereas a gallop in which the extra sound occurs between the second and first normal heart sounds is almost always a pathological finding and of grave significance, when the extra sound occurs between the first and second sounds, it is a benign phenomenon (mid-systolic click or gallop). It may be heard in some normal subjects, and when it accompanies organic disease, it does not add to the seriousness of the prognosis. There is a simple method of determining the fact that the sound is present in mid-systole. After identifying the three sounds at the apex of the heart, the stethoscope is rhythmically placed higher and higher over the præcordium. The middle one of the three sounds will be found to wane gradually and finally disappear on approaching the aortic

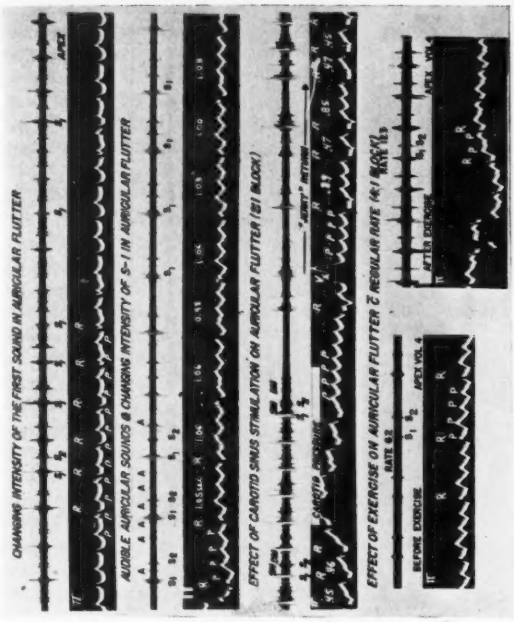
A
CHANGING INTENSITY OF THE FIRST SOUND IN AURICULAR FLUTTER
AUDIBLE AURICULAR SOUNDS @ CHANGING INTENSITY OF S–1 IN AURICULAR FLUTTER
B
EFFECT OF CAROTID SINUS STIMULATION ON AURICULAR FLUTTER (2:1 BLOCK)
C
EFFECT OF EXERCISE ON AURICULAR FLUTTER @ REGULAR RATE (4:1 BLOCK)
D

FIG. 9.—Four different cases of auricular flutter. (A) Upper tracing shows changing intensity first sound (S–1) with changing P–R relationships. Intensity of S–1 is not related to previous diastolic pause. (B) Second tracing shows changing S–1 (due to alteration in P–R interval) with almost regular ventricular rate. Note auricular sounds (a) even during systole. (C) Third tracing shows slowing of ventricular rate following carotid pressure with jerky return to original tachycardia. (D) Lowest tracings show a regular ventricular rate of 62 exactly doubled (123) after brief effort.

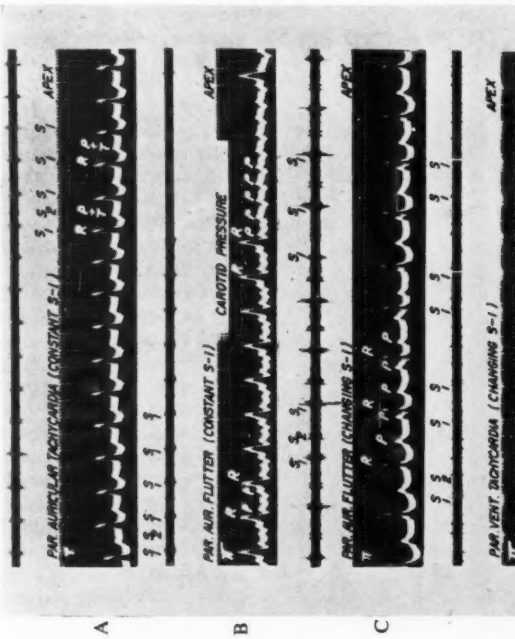

FIG. 8.—The first heart sound in various tachycardias. (A) Note constant intensity of first sound (S–1) in upper tracing (par. aur. tachycardia). (B) Second tracing shows constant S–1 with aur. flutter having regular 2:1 block. (C) Third tracing shows auricular flutter with slightly irregular ventricular rate with changing intensity of the first sound. (D) Fourth tracing (par. vent. tachycardia) shows changing S–1 with perfectly regular ventricular rate. (E) Lowest tracing shows changing first sound with auricular fibrillation.

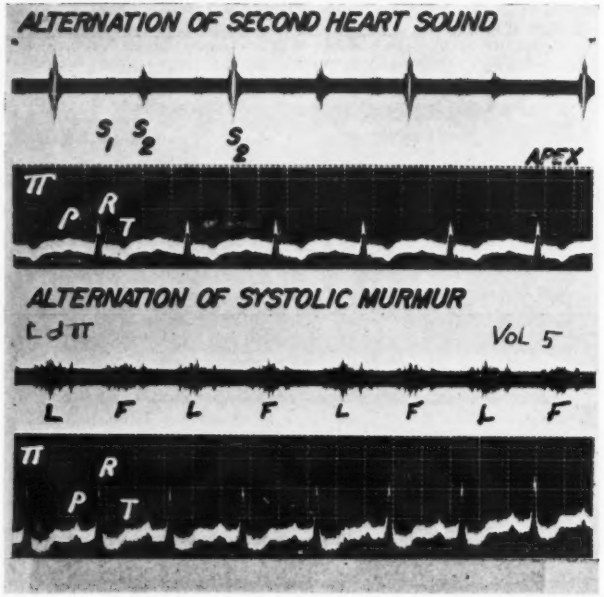

FIG. 10.—Upper tracing: Man, aged 61 years, with hypertensive heart disease. Note marked alternation of apical second sound (S–2). Lower tracing: Woman with calcific aortic stenosis. Note alternation of systolic murmur (SM). Loud = L. Faint = F.

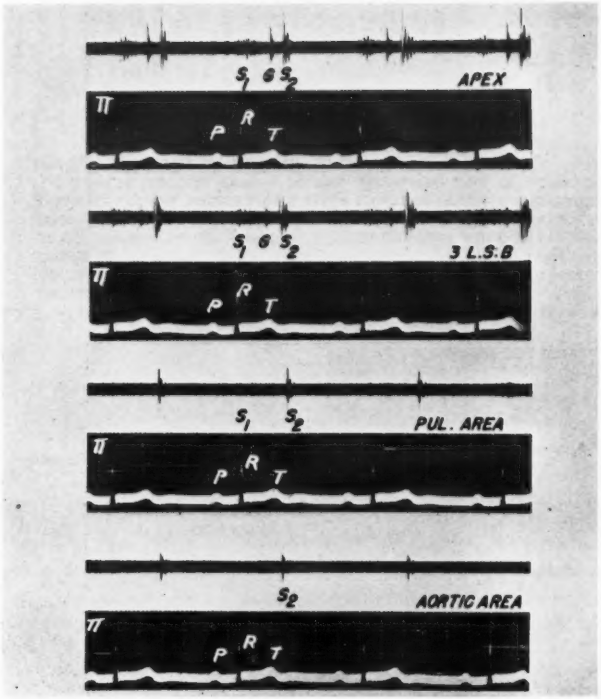

FIG. 11.—Systolic gallop disappearing at base. (Timing of gallop by "inching.") Woman, aged 26, with question of epilepsy. No organic heart disease. Note benign systolic gallop sound (G) occurs between first (S–1) and second sound (S–2), loudest at apex and gradually disappearing on reaching base. S–1 decreased because of long P–R (0·24 sec.).

area (Fig. 11). One will be left with the two normal sounds, making it clear that the sound that was audible at the apex and lost at the base of the heart occurred in mid-systole. This method of "inching" is helpful not only in identifying the types of gallop but also other events that occur in different parts of the cardiac cycle.

Systolic Murmurs

The interpretation and significance of a systolic murmur still remains a puzzling problem. It is quite true that many systolic murmurs (generally of slight intensity) are heard when there is no abnormality of the heart and, in fact, in those who have no structural disease whatever. In others, though there may be minor abnormalities, normal health and life expectancy is maintained. However, it is also true that many systolic murmurs detected in otherwise healthy people and regarded as benign or inconsequential, in later years have proved to be due to some structural abnormality. I look back at instances of this sort in which a congenital abnormality such as pulmonary stenosis or auricular septal defect was subsequently found by venous catheterization, which explained the "functional" basal systolic murmur. Others, ten or twenty years later, proved to have calcific aortic stenosis. Still others that had a slight apical systolic murmur developed unequivocal evidence of mitral stenosis or bacterial endocarditis. I believe it is fair to say that, whereas much too great emphasis was attached to the presence of a systolic murmur a few decades ago, too little attention has been paid to it in recent years.

It is not the purpose of this discussion to go into the mechanism or interpretation of the systolic murmur. However, I would urge that consideration be given to its detection and its possible significance, and that particular attention be paid to its intensity. Although faint systolic murmurs may be heard in otherwise healthy subjects, loud ones almost always denote some disease, generally of the cardiovascular apparatus. For this reason, I have become accustomed to grade the intensity of systolic murmurs from one to six. Grade one is the faintest and will generally not be audible until the examiner has listened for several cycles. Grade six is the murmur that is sufficiently loud to be heard with the stethoscope just removed from the chest wall. The intervening grades (2 to 5) are of intermediary intensity. Although at first glance this may seem a cumbersome terminology, it is surprising how quickly different observers will fall in line with each other, and rarely differ in their terminology by more than one gradation. The result of the use of such terminology is that one observer can more readily understand and compare the findings of another with his own. Furthermore, it enables the physician to divide systolic murmurs into those that are likely to be associated with organic disease (grade three or louder), and those that commonly are found without structural disease of the heart (grade one or two).

There appears to be certain misconceptions in our current teachings concerning the transmission of murmurs. There is a prevailing opinion that murmurs are transmitted by or with the blood stream. One frequently is told that a particular systolic murmur heard at the base of the heart is transmitted to the carotid artery, or another at the apex region is transmitted to the axilla. These observations are made as added evidence that the murmur in the first instance is due to aortic stenosis, and in the second instance is due to mitral insufficiency. There is now abundant proof that the transmission of murmurs is mainly a function of its intensity, and that bony structures are the best peripheral conductors. Very loud murmurs, no matter what their origin may be, are propagated in all directions, and may be well heard over the carotid arteries or in the axilla. A loud systolic murmur of Roger's disease or pulmonary stenosis may be well heard in the carotid arteries (Fig. 12). Here the flow of blood producing the turbulence is within the heart or chest and not out into the peripheral circulation, and yet the resultant murmur is transmitted to the neck. In fact, very loud murmurs (systolic or diastolic) are easily audible over the olecranon process of the elbow, even when arterial flow to the arm is completely occluded (Fig. 13). This is convincing evidence that the propagation of murmurs is through bones. The conclusions from these observations is that the murmur of aortic stenosis is transmitted to the neck because the point of maximum intensity of the murmur is near the neck, and that the mitral systolic murmur is well heard in the axilla because it is near the axilla.

Miscellaneous Observations

There are numerous other auscultatory findings of practical importance that can be easily elicited, which are beyond the scope of this discussion. Careful attention to the exact timing of the murmurs of mitral stenosis throws light on many otherwise baffling problems in diagnosis. The importance of the degree and duration of ventricular filling on the murmur of mitral stenosis has not been sufficiently emphasized. The presystolic murmur of mitral stenosis can disappear even when the auricles are contracting normally, if the diastolic interval is sufficiently long (Fig. 14). Not infrequently patients are observed who show no murmur whatever in

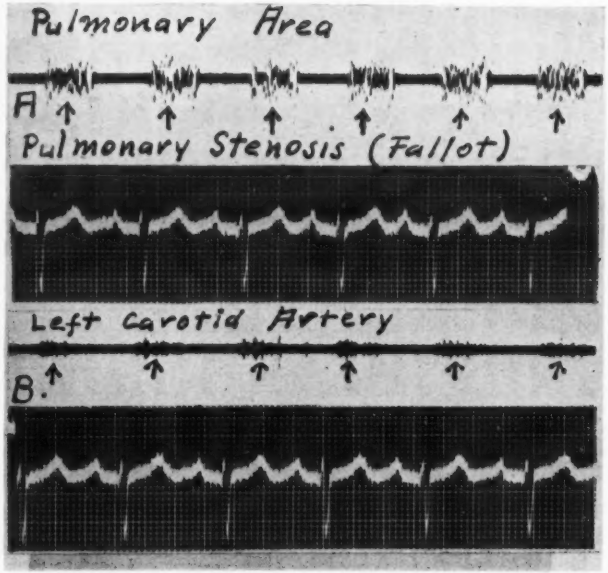

FIG. 12.—Woman, 29 years old, with tetralogy of Fallot. Upper tracing shows loud pulmonic systolic murmur. Lower tracing shows same systolic murmur over left carotid artery.

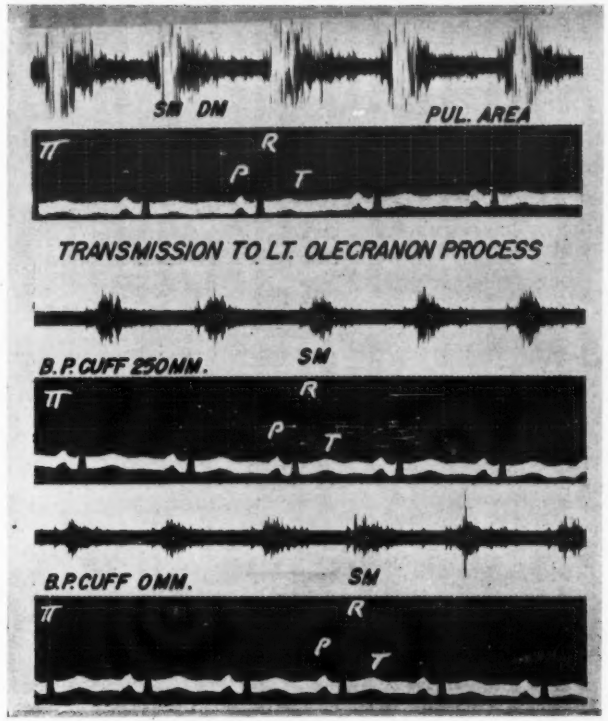

FIG. 13.—Murmur of patent ductus arteriosus. Woman, 23 years old, with patent ductus arteriosus. Note loud continuous murmur in upper tracing from pulmonic area. Middle strip shows systolic murmur (SM) at olecranon process with blood pressure cuff inflated to 250 mm. Same murmur readily heard at elbow with cuff uninflated (lower tracing).

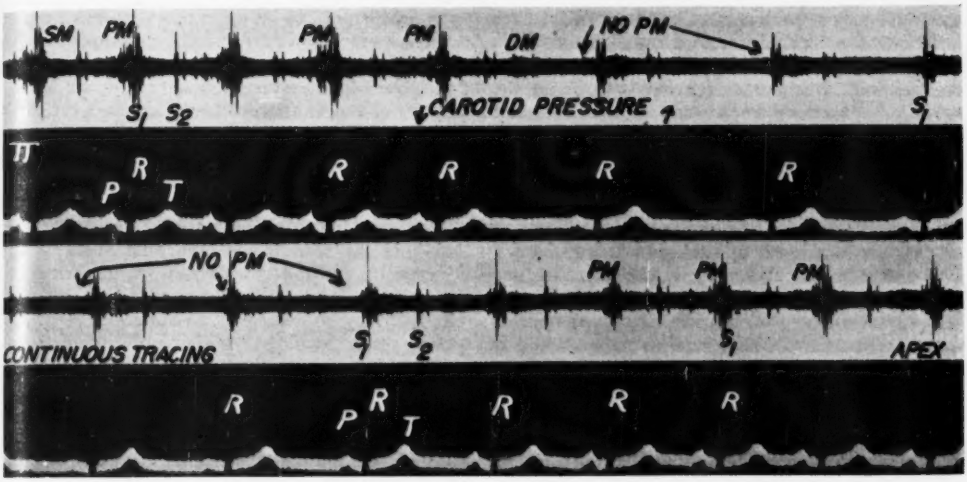

FIG. 14.—Effect of long diastole on presystolic murmur of mitral stenosis. Woman, 26 years old. Diagnosis mitral stenosis. Note classical presystolic murmur (PM) disappears with long diastolic pauses following carotid pressure.

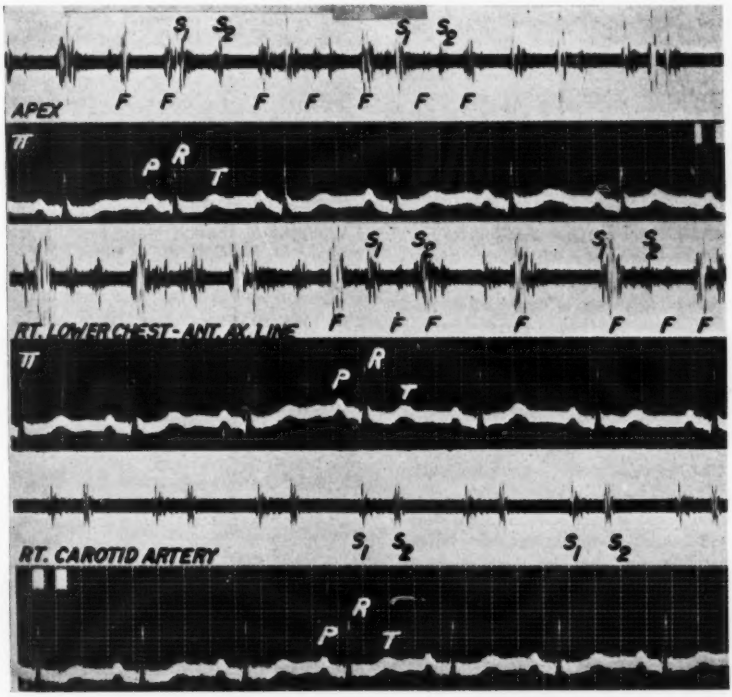

FIG. 15.—Diaphragmatic flutter. Woman 32 years old with mitral stenosis and diaphragmatic flutter. Upper tracing from apical region shows numerous sounds, a combination of normal heart sounds (S–1, S–2) and flutter sounds (F). This was at first clinically interpreted as auricular fibrillation. Note that flutter sounds (F) were very prominent in right axilla (middle tracing) and were entirely absent over right carotid artery.

diastole on most careful auscultation, and yet have unequivocal evidence of mitral stenosis, as shown by the finding of a calcified valve on fluoroscopic examination. Such cases are likely to be in the advanced stages of heart failure with auricular fibrillation. The absence of a diastolic murmur is then probably the result of the very slow velocity of blood flow and the large volume of residual blood in the various chambers of the heart.

Just to mention an occasional reward the physician may obtain as a result of intelligent auscultation, I may mention the following experience. In this particular patient who had obvious evidence of mitral stenosis, on one occasion very peculiar heart sounds were heard. They seemed baffling, and it first made one think that auricular fibrillation was present. On listening over different parts of the chest, it quickly became clear that regular sounds at a rate of about 120 could be heard far removed from the heart, even in the right axilla (Fig. 15). They were entirely independent of the pulse which was also regular, but 75 to the minute. At other times, these aberrant sounds were absent. The tentative diagnosis of diaphragmatic flutter was made. This was confirmed by fluoroscopic examination, at which time regular rapid contractions of the diaphragm could readily be seen.

I hope I have commented sufficiently on various aspects of auscultation to lend some support to the proposition that this method of examination is valuable and simple. I also have reason to believe that much of the knowledge that is at present available is not being utilized to the full. I may urge that greater emphasis be placed on this subject in our medical schools, so that this valuable and inexpensive method of diagnosis be not neglected. It is also hoped that further interest and study will clarify many problems in auscultation at present poorly understood.

Finally, I wish to thank my British friends and physicians for the privilege and honour of coming here to address you. Many of the leading students of heart disease in the United States obtained their early inspiration and training here in your country, under your great teachers. This will always remain a debt quite difficult to repay. But in a broader sense there are no debts or credits for the medical profession is truly universal in its brotherhood.

References

Dock, W. (1933). *Arch. intern. Med.*, **51**, 737.

Gee, S. J. (1908). *Auscultation and Percussion*, 6th ed., Oxford Medical Publication, London, p. 84.

Griffith, T. W. (1912). *Heart*, **3**, 143.

Harvey, W. P., and Levine, S. A. (1948). *Amer. Heart J.*, **35**, 924.

Henderson, Y., and Johnson, E. (1912). *Heart*, **4**, 69.

Levine, S. A. (1927). *Amer. Heart J.*, **3**, 177.

Lewis, T. (1915). *Lectures on the Heart*, Paul B. Hoeber, 1915, p. 60.

—— (1925). *The Mechanism and Graphic Registration of the Heart Beat*, 3rd ed., Shaw & Sons, Ltd., p. 181.

Strazhesko, N. D. (1906). *Russ. Vrach. S. Peterb.*, **5**, 603.

Wolferth, C. C., and Margolis, A. (1930). *Arch. intern. Med.*, **46**, 1048.

HEART-VECTOR AND LEADS. PART III

GEOMETRICAL REPRESENTATION

BY

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Received May 31, 1948

In 1947 Wilson, Johnston, and Kossmann gave a generalization of the triangle-rule of Einthoven. In this paper they describe the use of a tetrahedron; their method is based on the same assumption as Einthoven's. We have tried to give a rational description of the relation between heart-vector and leads, based on firmly established physical principles (Burger and van Milaan, 1947).

It is not self-evident that the electric action of the heart can be represented by *one* heart-vector. This is only correct if one is allowed to consider the dimensions of the heart as very small compared with those of the trunk. As a matter of course this *can* only be a useful approximation if the electrodes are applied far from the heart. In that case it does not matter very much whether the electric action originates in the one or in the other part of the heart muscle. As can be shown by mathematical analysis, the electric action of the heart can then be represented by *one* vector, acting in the " centre " of the heart. But for præcordial leads, i.e. electrodes near the heart, this simplification is certainly not entirely correct, and we shall return to this point at the end of this paper.

The formulation of the relation between heart-vector and leads can be based on well-founded physical laws. It is then possible to say with certainty what is known *à priori* and what must be found by *measurement*. The linear relation between

$$Lead = aX + bY + cZ \quad . \quad . \quad . \quad . \quad (1)$$

each lead and the rectangular components X, Y, Z of the heart-vector is certain.

We have related these components to a "natural" co-ordinate-system, of which the *Z*-axis is parallel to the axis of the body and of which the *Y*-axis is perpendicular to the frontal plane (Burger and van Milaan, 1946, 1947).

As we have explained in the papers mentioned, the coefficients *a*, *b*, *c*, which are independent of the heart-vector, must be determined experimentally. For this one can use a phantom or measure on a cadaver. We have done the former ourselves (l.c.): the latter method is used by others (Fahr and Weber, 1915; de Waart and Storm, 1935; Storm, 1936; Wilson *et al.*, 1947. But whichever method is used one must proceed objectively and leave the decision to the experiments. We have, therefore, provisionally accepted the values of the coefficients *a*, *b*, *c*, for a number of leads as the experiments gave them. By repeating the measurement and by varying the conditions we have tried to improve on their reliability.

With our phantom we have tried to reproduce the human body as closely as possible but are fully aware of the numerical imperfection of the result. We took as a basis our measurements of the electric conductivity of the living human body (Burger and van Milaan, 1943). In these measurements we have not noticed the influence of membranes that by their high resistance could determine the course of the electric current. Nor did preliminary measurements on (dead) membranes give a high value for this resistance either.

From various sides other systems are proposed instead of the Einthoven triangle, all of them rectangular. In these cases the electrodes are applied on the body in such a way, that the lines connecting them are mutually perpendicular and parallel to the axes of the above mentioned "natural" co-ordinate system (Schellong, 1939; Sulzer and Duchosal, 1945; Trocmé, 1946). As an argument in its favour it is sometimes put forward, that it is simpler, but it is not always realized that these systems are just as arbitrary as the equilateral triangle of Einthoven. Measurements on a phantom indicate that from a quantitative standpoint, these systems do not improve matters. In our opinion the lucky hit of Einthoven comes nearer to

the true state of affairs than the rectangular systems mentioned above.

From some sides objections are raised against phantom measurements. Indeed we shall appreciate every suggestion for improvements on our phantom. Taking into account the internal structure of the human body, this might be a rational way to approach the truth nearer and nearer as regards the numerical values of the coefficients a, b, c.

The attempts made by physicians to give the relation between heart-vector and leads have always led to geometrical representations (triangle rule, rectangular systems, equilateral tetrahedron). From these representations the analytical conclusions are drawn only in the second place and the result is given as a formula. This, however, is a non-physical line of thought. The physical laws, governing the electric current density in the trunk as a consequence of the heart action, are of an analytical nature and this is also the case with their general consequence equation (1). One can proceed from this equation and it suffices in practice to find the heart-vector.

It cannot be denied, however, that a geometrical representation can be useful for the sake of an easy survey, but it has to be based on equation (1). Mathematically one has to derive from (1) the *general* geometrical representation, which is equivalent to this equation.

Those, who are acquainted with the vector calculus can deduce this representation from (1). The lead (=potential difference) is a scalar (undirected) quantity, i.e. a quantity characterized by only one single number, independent of the choice of the direction of the co-ordinate axes.* The quantities X, Y, Z are vector components determining together the heart-vector. The value of each of these quantities depends on the choice of the direction of the axes. With another choice of the axes, therefore, we have three other components X', Y', Z'. Their resultant, however, is the same heart-vector. Choosing other directions of the axes, the components X, Y, Z will vary, while $aX+bY+cZ$ must remain the same. Therefore, a, b, c must vary too, i.e. depend on the direction of the axes. It can be proved that they are the components of a vector $\overrightarrow{(a, b, c)}$. The relation between this vector $\overrightarrow{(a, b, c)}$, the heart-vector $\overrightarrow{(X, Y, Z)}$ (or $\vec{H}$) and the scalar lead can be represented geometrically. The lead is the product of the projection of $\vec{H}$ on $\overrightarrow{(a, b, c)}$ and the length $\overrightarrow{(a, b, c)}$ of this last vector. φ being the angle between the two vectors, this relation can be denoted in a more symmetrical form:

$$\text{Lead} = \vec{H} \,.\, \overrightarrow{(a, b, c)} \cos\varphi \quad . \quad . \quad . \quad (2)$$

This formula is analogous to the well-known relation:

$$work = force \times displacement \times \cos\varphi$$

The work is the scalar product of force and displacement just as in our case the lead is the scalar product of the vector $\overrightarrow{(a, b, c)}$ and heart-vector $\vec{H}$.

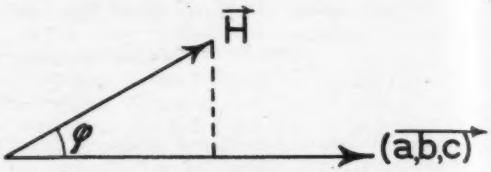

FIG. 1.—A lead is the product of the projection of the heart-vector $\vec{H}$ on the vector $\overrightarrow{(a,b,c)}$ and the length of the latter.

To determine X, Y, Z (i.e. $\vec{H}$) we need three equations of the type (1) and, therefore, three leads (mutually independent). For simplicity we shall suppose that they have one electrode in common, for which as a rule the left leg (F) is taken. If the potential differences between three electrodes and F are known, we have three equations (1) to solve the three unknowns X, Y, Z. Interpreted geometrically, this means that there are three vectors $\overrightarrow{(a, b, c)}$, originating in a common point, corresponding to F (Fig. 2). The end points of these vectors correspond to the other three electrodes 1, 2, and 3, the position of which can be chosen arbitrarily. The points F 1 2 3 form a non-equilateral tetrahedron, the shape of which depends on the position of the electrodes on the body. The position of the tetrahedron, i.e. the direction of its edges with respect to the body is fixed, but it is obvious that its place may be chosen arbitrarily.

This tetrahedron makes it possible to represent geometrically the relation between heart-vector and leads. In order to determine lead (12), for example, the heart-vector must be projected on the edge (12) and the projection must be multiplied by the length of this edge (formula 2).

For a description of a general relation between heart-vector and leads a restriction to the three points 1, 2, 3 is not allowed. With each point on the surface of our body corresponds a point in the space of the tetrahedron F 1 2 3. All those points, 4, 5, 6, together form a surface, on which are also situated the points F 1 2 3. An arbitrary point, say 10, can

* Other examples of scalar quantities are: temperature, mass, time.

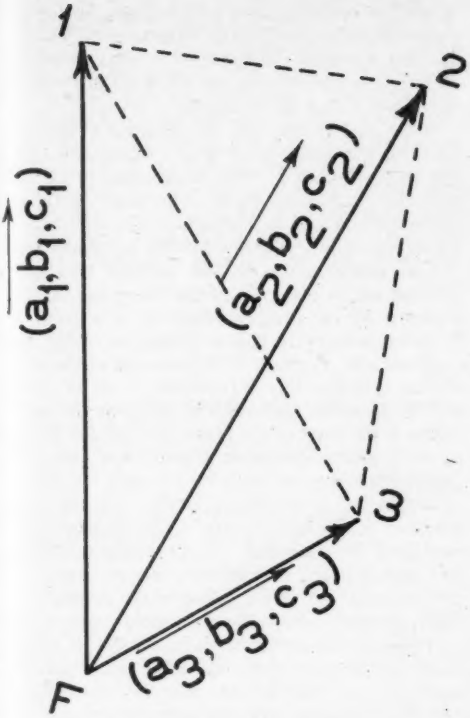

FIG. 2.—Three vectors $\overrightarrow{(a_1b_1c_1)}$ $\overrightarrow{(a_2b_2c_2)}$ and $\overrightarrow{(a_3b_3c_3)}$ determining a tetrahedron with which the relation between the heart-vector and three leads can be described.

be determined with the aid of the phantom in the following way. In this phantom an electrode is applied in a position corresponding to the position of the electrode 10 on the body of the patient. On the phantom lead 10 *F*, i.e. the potential difference of the electrodes 10 and *F*, arising from the known components *X*, *Y*, *Z* of the vector of the artificial heart, is measured. The proportionality of actors being a_{10},b_{10},c_{10}, the lead is given by

$$Lead\ 10\ F=a_{10}X+b_{10}Y+c_{10}Z.$$

In the space of the tetrahedron *F* 1 2 3 the vector $\overrightarrow{a_{10},b_{10},c_{10}}$ is drawn, the end-point of which has the co-ordinates a_{10},b_{10},c_{10}. This point is the "image" of the position of the electrode 10 on the surface of the body of the patient. The above mentioned surface, formed by all image-points (Fig. 3) can be used as follows, to find the relation between the heart-vector and any lead. Let *p* and *q* be two arbitrary positions of electrodes on the surface of the

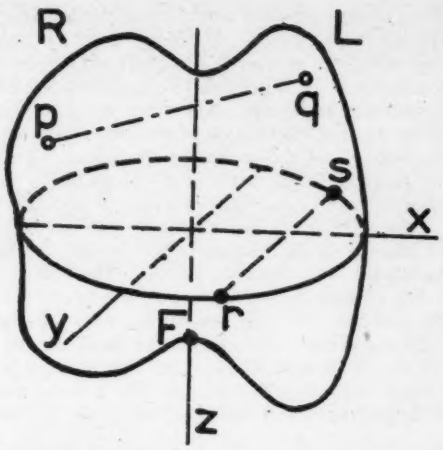

FIG. 3.—Surface in the image-space, corresponding to the surface of the human body. *X*, *Y*, *Z*=co-ordinate axes, *r* and *s*=images of two electrodes, which give a potential difference, depending on the *Y*-component of the heart-vector only.

body. These give on the surface of Fig. 3 the "image-points" *p*, *q*. Now, lead *pq* equals the product of the projection of the heart-vector, drawn in the correct direction in the image-space, on the line *pq* and the length of this line (formula 2). It is possible to reverse this process and to deduce in a way, which we shall not explain here, the heart-vector from three leads, the image-surface of Fig. 3 being given.

An electrode within the body has also an image-point which can be found with the aid of the phantom. Such points are situated in the image-space outside the surface of Fig. 3 so that, the nearer the electrode inside the human body is to the heart, the farther away they lie.

In a preceding paper (Burger and van Milaan, 1947) pairs of electrodes on the surface of the human body are mentioned, giving a potential difference that depends on one component of the heart-vector only. Then in equation (1) two coefficients are zero e.g. *Lead*=*aX*. The geometrical representation enables us to take a simple view of the problem. If a lead for example has to be found depending only on the postero-anterior component *Y* of the heart-vector, the projections of the *X* and *Z* component on the line joining the image-points of the electrodes must be zero. This line, therefore, *in the image* must be parallel to the *Y*-axis. In Fig. 3, *r* and *s* are the images of electrodes, fulfilling this requirement. The relation between the position of the electrodes and the image-point

being known, the place where the electrodes must be applied is determined. It follows from the figure, that an infinite number of combinations *rs* meet the requirement. It is possible to choose one electrode for instance arbitrarily. The other one (*s*) is found as the point of intersection of the line *rs* (parallel to the *Y*-axis) and the surface of the image. To use this method efficiently a practical combination must be chosen out of the infinite number of possibilities.

This geometrical representation can serve also for elucidating the meaning of the central terminal, introduced by Wilson (1935). This electrode is not applied on the surface of the body, but is the junction *CT* (Fig. 4) of the ends of three equal resistances connected to the electrodes *R*, *L*, and *F*. If these resistances are high enough, it is easily proved, that the potential of *CT* is the mean of the potentials of the electrodes *R*, *L*, and *F*. As it is not the potential itself that matters, but only the potential differences, it is formulated better by saying that the potential difference of *CT* and an arbitrary electrode is the mean of the potential differences of *R*, *L*, and *F* and this electrode.

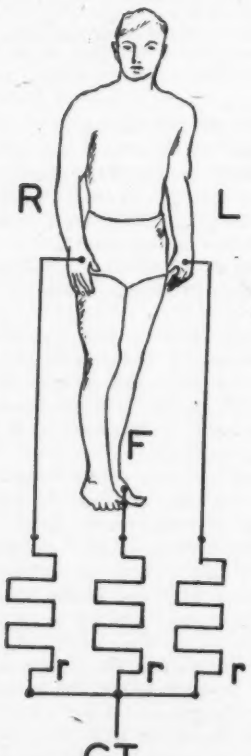

FIG. 4.—The central terminal *CT* is the junction of the ends of three equal resistances *r*, connected to the electrodes *R*, *L*, and *F*.

It can be proved, that it is possible to indicate a point in the image-space (Fig. 3) corresponding to the central terminal. This image-point is the centre of gravity of triangle *RLF*. This point can be used for the geometrical determination of leads just as was explained above for points on the image-surface.

The image-point of the central terminal is situated inside that part of the image-space, that is enclosed by the image-surface. It is a point, that does not possess the special properties of the upper or lower side, the right or the left side of the image-surface. Inside this it occupies a more or less central position, ignoring the fact that the surface image at the front of the plane *RLF* (and of *CT*) has a much greater extension than at the back. The image has a pigeon breast.

Interpreted geometrically in this way, the central position of Wilson's central terminal seems to be explained satisfactorily. A more exact definition of a central point, in which not only the points *RLF* are involved, but all the points of the surface of the body, is possible but seems to be hardly worth while.

It may be asked whether it is possible to obtain a point of mean potential with three electrodes other than *RLF* in such a way, that this point has no potential difference with the central terminal. This can be realized fairly approximately by experiment (personal communication by J. B. Kleyn). It can moreover be deduced from our geometrical considerations, that this can be obtained exactly and even in an infinite number of different ways. For there are an infinite number of triangles in the image-space, of which the angular points are situated on the image-surface and of which the centre of gravity coincides with the image-point of the central terminal. This, however, does not hold good for the image of the central terminal only, but for every point inside the image-surface.

In the preceding it has always been supposed, that the dimensions of the heart are very small compared with those of the trunk. In reality this is not the case so that the image-surface will depend on the position of that part of the heart muscle, of which the action is being studied. Comparing diametrically situated parts of the heart-muscle (e.g. heart point and heart base) this difference is appreciable as revealed by measurements on a phantom. But on the average the distances are much smaller so that in a first approximation this effect may be neglected. This, however, holds good only if the electrodes are applied at a rather large distance from

the heart, as is the case for *R*, *L*, and *F*. A præcordial electrode, however, will be situated too near to the heart. The part of the heart muscle near the thoracic wall will contribute to the præcordial lead relatively more than the other parts. It is true that this reduces the value of the preceding considerations. But on the other hand it may enable one to find the place of disturbances in different parts of the heart muscle. This is understood quite well by physicians and it is for this very purpose that the præcordial leads are recommended. But the relation between the place of the defect and the electrocardiogram is determined in an empirical way only. A more correct comprehension of this relation is only to be obtained by a generalization of the exact physical treatment of the problem.

Summary

The relation between heart-vector and leads can be represented geometrically. This representation is a generalization of Einthoven's triangle. It is possible to elucidate the meaning of the central terminal, proposed by Wilson.

References

Burger, H. C., and van Milaan, J. B. (1943). *Acta med. scand.*, **114**, 584.

——, —— (1946). *Brit. Heart J.*, **8**, 157.

——, —— (1947). *Ibid.*, **9**, 154.

Fahr, G., and Weber, A. (1915). *Dtsch. Arch. Klin. Med.*, 117.

Schellong, F. (1939). *Grundzüge einer klinischen Vektordiagraphie des Herzen*, Berlin, Springer.

Storm, C. J., (1936). *Thesis*, Batavia.

Sulzer, R., and Duchosal, P. W. (1945). *Cardiologia*, IX, Fasc. **1/2**, 106.

Trocmé, Ch. (1946). *Arch. Mal. Coeur*, **39**, 80.

de Waart, A., and Storm, C. J., (1935). *Arch. Néerl. Physiol.* **20**, 255.

Wilson, F. N., Johnston, F. D., Macleod, A. G., and Barker, P. S. (1935). *Amer. Heart J.* **9**, 447.

——, ——, and Kossmann, C. F. (1947). *Ibid.*, **33**, 594.

VENTRICULAR ESCAPE IN ACUTE RHEUMATISM

BY

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Received July 20, 1948

Ventricular escape can be produced by depression of the sino-auricular node as a result of digitalis therapy, marked sinus arrhythmia, sinus bradycardia, or pressure on a hyperirritable carotid sinus, or by hyperirritability of the auriculo-ventricular node. The latter type is the one referred to here. It is differentiated from auriculo-ventricular dissociation due to complete heart block by the fact that the ventricles beat at the same or a more rapid rate than the auricles. There is no general agreement about the ultimate mechanism of its production. The most likely and usually accepted cause is irritation of the node by adjacent pathological changes, such as transient œdema around a rheumatic nodule or cellular infiltration of the node with or without endarteritis in the small vessels supplying it (Gross and Fried, 1936; quoted by Stein and Bartlett, 1946). Stimulation of the sympathetic or paralysis of the vagus supply to the node consequent upon the same rheumatic process could have a similar effect. There is also no agreement as to its significance in prognosis. Dressler (1930) found that it occurs during the course of acute articular rheumatism. Stein and Bartlett (1946) state that it must not be regarded as an expression of the natural cardiac mechanism, that it is transient, and that it probably bears no relation to the outcome of acute rheumatism. They state, however, that once a complete heart block has been excluded it is reasonable to give a good prognosis. Wendkos and Noll (1944) had previously arrived at the same conclusion. Cutts (1937) does not concur but agrees with Richardson (1915) that auriculo-ventricular dissociation due to ventricular escape is not of itself serious but that it is frequently associated with severe infection or severe and chronic heart disease.

Two cases of acute rheumatism have recently been seen at Alder Hey Hospital exhibiting ventricular escape.

Case Notes

Case 1. This patient was a girl of 11 years who had suffered with recurrent attacks of tonsillitis until the age of 7, when her tonsils were removed. From that time until six days before admission to hospital she had no further attacks. She was sent home from school complaining of a sore throat and pain in the back and thighs. On the day of admission the pain in the legs, particularly the knees, was severe enough to make her cry if moved.

On examination the temperature was 99·4° F., and the pulse rate 128 a minute. She was thin, pale, and apprehensive; there was no dyspnœa nor cyanosis. The throat was slightly injected with no visible tonsillar tissue and no membrane. The tonsillar glands were a little enlarged and tender. Both knees were painful on movement, which was limited to 45°, and tender to touch. There was no redness or swelling and rheumatic nodules were not found. The pulse was generally regular with occasional periods of irregularity. The heart apex beat was in the fifth left intercostal space, 6 cm. from the midline, the heart sounds were all softened and there were no murmurs. Auscultation revealed the same cardiac arrhythmia as the pulse, which was considered to be due to extrasystoles.

The condition was diagnosed as acute rheumatism and treatment was begun with sodium salicylate, 15 grains, and sodium bicarbonate, 20 grains, four-hourly; this was continued for four weeks.

On the day following admission pain in the knees was less, but she complained of a dull præcordial pain which was eased by cataplasma kaolin. The cardiac rhythm was regular; no change had taken place in the heart sounds. Two days after admission the pain in the legs and præcordium had gone; and her general condition had improved. The cardiac rhythm, however, was usually irregular except for brief periods. The rate was still 110 to 130 a minute; in spite of this rapid rate the irregularity was typical of extrasystoles. A cardiogram showed auriculo-ventricular dissociation, auricular rate 83 a minute, ventricular rate 107 a minute

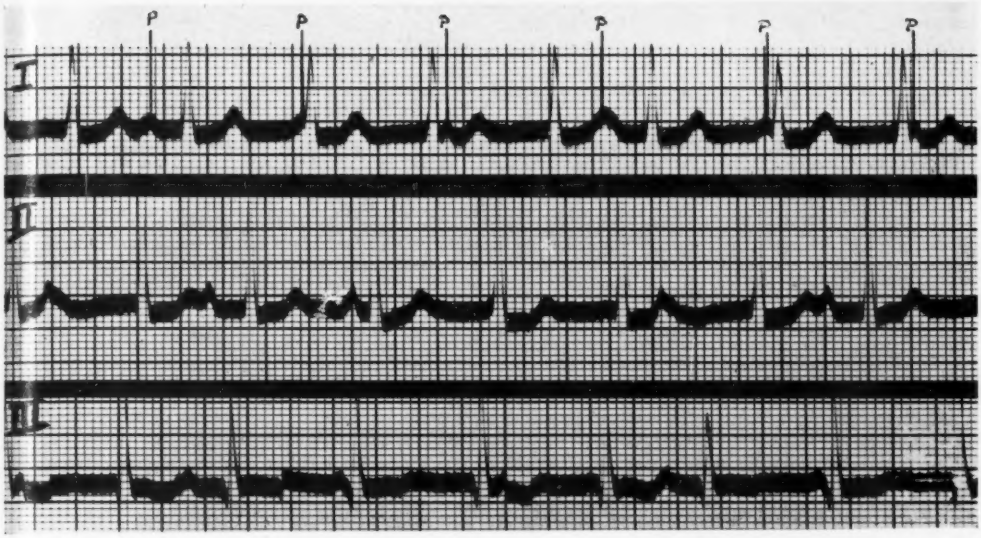

FIG. 1.—Case. 1. Ventricular escape. Auricular rate 83 a minute; ventricular rate 107 a minute.

(Fig. 1). On the fourth day the cardiac rhythm was quite regular at a rate of 92 a minute. The first apical sound was softened but the others were now clear and much louder. The general condition continued to improve and by the end of another week the quality of all the heart sounds was good, but a soft localized apical systolic murmur had become evident. Five weeks after admission, cardiac irregularity typical of extrasystoles was again noted, the rate being 75 a minute. This was the only occasion on which irregularity had been found since the second day after admission. A cardiogram showed sinus arrythmia and two dissimilar ventricular extrasystoles. At the end of the sixth week in hospital she was regarded as convalescent and five weeks later she was discharged. The heart sounds were normal but the apical systolic murmur, still soft and localized, persisted. The cardiogram was normal. The E.S.R., which had been 92 mm. in the first hour (Westergren) on admission, was now 5 mm., and X-ray examination showed a normal cardiac outline.

Case 2. A girl, aged 8 years. Apart from scarlet fever at the age of five she had been well until 7 days before admission when she complained of severe pain behind both knees and was unable to walk. The pain subsided after 24 hours. For the next six days she complained of stiff knees on waking up in the mornings and occasional fleeting pains during the day. She was feverish for two to three days before admission.

On examination the temperature was 101° F., and the pulse rate 124 a minute. She was flushed and the skin was hot and dry. There was a fading erythema marginatum on the front of the chest and abdomen. The tongue was furred but the throat appeared healthy. The movements of the knee joints were full and there was no swelling or redness. The pulse was regular, the heart apex beat in the fourth left intercostal space 6 cm. from the midline, the pulmonary second sound accentuated, and there was a soft blowing apical and basal systolic murmur. No abnormality was found in the lungs or abdomen, and there were no rheumatic nodules though a few were found late in the illness on the wrists and elbows. A diagnosis of acute rheumatism with rheumatic carditis was made and treatment with calcium aspirin, 10 grains, t.i.d., was begun.

The temperature settled after two days and the sleeping pulse rate fell to about 80 a minute. X-ray examination showed the cardiac outline to be normal and the E.S.R. was 79 mm. (Westergren). Progress was good for 4 weeks but choreiform movements, which continued for 2 weeks, were then noticed, and an aortic diastolic murmur was heard. The blood pressure in the arm was 100/70 mm. Calcium aspirin was replaced by sodium salicylate, 15 grains, and sodium bicarbonate, 20 grains, four-hourly.

Eight weeks after admission to hospital, the apex beat was located in the fifth left intercostal space 9 cm. from the midline, and a late diastolic murmur of low rumbling quality was heard at the apex. The child was pale and apathetic during the succeeding 2 weeks then the temperature which had been normal for 8 weeks rose to 102·4° F., but settled again after 3 days. The pulse rate which had remained steady below 90 a minute increased to 120 to 130 a minute and the heart apex beat had become diffuse and tumultuous, 10 cm. from the midline. A fleeting scratchy pericardial friction rub was audible in the second left interspace adjacent to the sternum; this was heard on and off for the next 7 days. The erythrocyte sedimentation rate was 83 mm. and an X-ray examination now showed marked generalized cardiac enlargement. The pulse rate continued between 120 and 130 a minute, and the blood pressure 105/20 mm. Her general condition improved somewhat and remained so for another three weeks. She then suddenly became dyspnœic and ashen grey in colour. The pulse rate rose to around 150 a minute, the rhythm being grossly irregular. Auricular fibrillation was diagnosed but a cardiogram showed A-V dissociation with regular auricular rhythm at a rate of 150 a minute. The ventricular rate was approximately the same with occasional slowing. At these times the sino-auricular node appeared to take charge of the ventricle, the P–R interval being 0·2 sec. (Fig. 2). Digoxin was administered for 48 hours. On the next day slight pretibial œdema occurred and the liver had become enlarged to two fingers' breadth below the costal margin and was very tender. The pulse rate had dropped to 100 a minute, the rhythm still being irregular, but the character of the irregularity had changed to one in which every sixth beat was dropped. Auscultation revealed a similar rhythm but whereas every sixth beat was missing at the pulse the normal heart sound was replaced at these times by a solitary faint clicking sound, only audible at the base. Later in the day the heart rate rose to between 130 and 140 a minute but otherwise no change occurred. On the following day the heart rate was regular but a loud pericardial friction rub was heard at the base of the heart. The liver was a little smaller and not so tender. A cardiogram showed that the rhythm was normal at the rate of 136 a minute. The P–R interval was prolonged to 0·2 sec. (Fig. 3). A week passed, her general condition was very poor but remained stationary. The cardiac rhythm was found to be irregular again with every sixth beat missing; rate 140 to 150 a minute. Auscultation revealed the same clicking sound

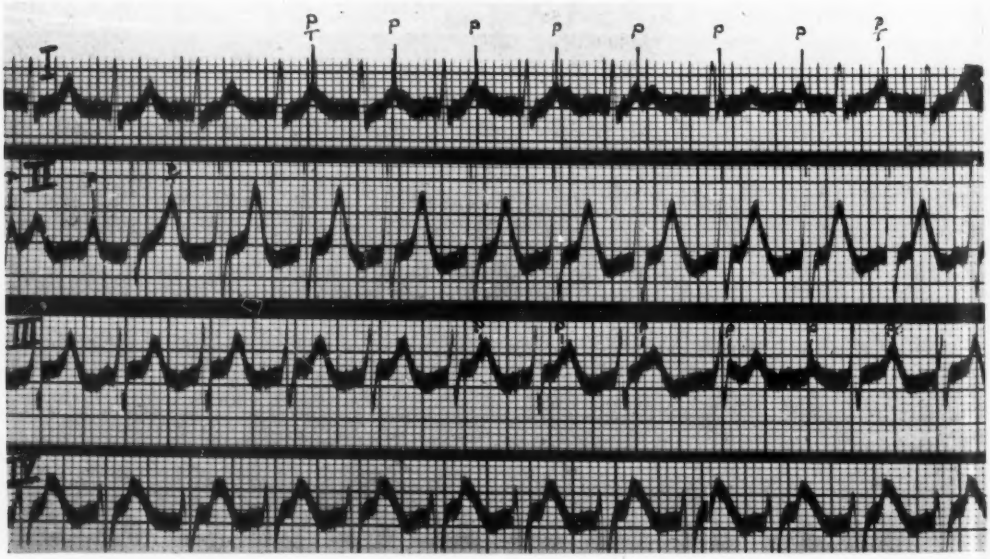

FIG. 2.—Case 2. Ventricular escape. Auricular rate 150 a minute; ventricular rate approximately the same with occasional slowing. When this occurs an isolated complex results which originates in the sino-auricular node the P–R interval being 0·2 sec.

FIG. 3.—Case 2. Normal sinus rhythm. P–R interval 0·2 sec.

replacing every sixth normal beat. The pericardial friction rub could now be plainly heard all over the præcordium. On the next morning she collapsed and died in 15 minutes.

Necropsy. Moderate wasting.

Heart. Marked fibrinous pericarditis with but a small amount of fluid present. There was dilatation of both ventricles with softening of the myocardium. The mitral valve showed typical rheumatic vegetations along the line of closure, a " MacCallum patch " was present and a more generalized roughening of the endocardium of the left ventricle towards the apex. The aortic valve was incompetent with recent rheumatic vegetations on the cusps. Tricuspid and pulmonary valves appeared normal. No evidence of previous rheumatic valvulitis.

The *lungs* showed a few subpleural petechiæ only.

The *liver* was enlarged and congested, the cut surface showed " nutmeg " change.

The *spleen* was enlarged by one and a half times normal size, the pulp firm with prominent follicles.

The *kidneys* showed no gross abnormality.

Other abdominal and pelvic viscera were normal.

THE DIAGNOSIS OF VENTRICULAR ESCAPE

In the series of cases reported there would appear to be no specific signs or symptoms that could be attributed to ventricular escape alone. White (1916) noted that one of his patients complained of palpitation and he found a cardiac irregularity that he ascribed to auricular extrasystoles. Wilson (1915) had three patients who complained of intense palpitation during experimentally produced A-V dissociation. Cutts (1937) mentions only one of his twelve cases as having subjective symptoms (" a jumping feeling in her chest ") but a number of them had unspecified cardiac irregularities. Five of the twelve had a first heart sound that varied from being softened to loud and snapping. Stein and Bartlett (1946) hold that a diagnosis cannot be made on clinical grounds, although auscultation may reveal an irregularity of rhythm similar to premature contractions or second degree heart block. There may be no irregularity recognizable clinically. Sudden rapid pulsation of the neck veins is said to be visible on occasion. All the cases described in association with acute rheumatism would appear to have developed ventricular escape early in the course of the illness.

The first described here had no symptoms and her arrhythmia was thought to be due to extrasystoles.

The second case was most unusual because, firstly, the ventricular rate was much more rapid than in others described (generally it is between 80 and 110 a minute), and the irregularity was therefore taken to be due to auricular fibrillation; and, secondly, there was acute cardiac failure with dyspnœa, œdema, and a rapidly enlarging, tender liver.

The discovery of an irregular rhythm with or without subjective symptoms; especially if associated

with a varying quality of the first heart sound and occurring in the course of acute rheumatism, usually early, should lead one to consider the possibility of ventricular escape and have an electrocardiogram recorded.

The Significance of Ventricular Escape in Acute Rheumatism

It seems generally agreed that ventricular escape occurring early in the course of acute rheumatism is transient and not in itself serious; and as all the cases with rheumatic carditis in the series quoted seem to have recovered in two months or so, it would not seem to indicate severe rheumatic carditis. Case 1 further supports this view. The ventricular escape in Case 2, however, occurring late in the course of severe rheumatic pancarditis, may have been responsible for the onset of acute cardiac failure. Furthermore although the second cardiogram taken when the rhythm was clinically regular showed no A-V dissociation, the subsequent clinical findings up to the time of death would suggest that it recurred. It is therefore suggested that in this particular case death was almost certainly due to severe pancarditis, though the A-V dissociation due to the ventricular escape may possibly have hastened the end.

Further electrocardiographic studies in cases of severe rheumatic carditis with cardiac failure, even when the rhythm is clinically regular might reveal that ventricular escape is more common and of more serious import than at present supposed.

Summary

Two cases of ventricular escape occurring during the course of acute rheumatism are described. One occurred early in the illness and is considered to be of little significance. The other occurred late in the course of severe pancarditis and it may have been responsible for the onset of acute cardiac failure. The auricular and ventricular rates in this case were unusually rapid.

I wish to thank Professor N. B. Capon and Dr. E. Noble Chamberlain for their helpful advice and criticism during the preparation of this paper; also Dr. W. E. Crosbie, Medical Superintendent of Alder Hey Children's Hospital for permission to publish these cases.

References

Cutts, F. B. (1937). *Amer. Heart J.*, **13**, 453.
Dressler, W. (1930). *Wien. Arch. inn. Med.*, **19**, 611.
Gross, L., and Fried, B. M. (1936). *Amer. J. Path.*, **12**, 31.
Richardson, H. B. (1915). *Arch. intern. Med.*, **16**, 517.
Wendkos, M. H., and Noll, J. (1944). *Med. Clin. North America*, **28**, 124.
White, P. D. (1916). *Arch. intern. Med.*, **18**, 244.
Wilson, F. N. (1915). *Ibid.*, **16**, 989.
Stein, I., and Bartlett, A. G. (1946). *Amer. J. med. Sci.*, **211**, 686.

DISSECTING ANEURYSM OF THE INTERVENTRICULAR SEPTUM*

BY

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Received July 22, 1948

Bernheim (1910, 1915) first drew attention to the occurrence of right heart failure as a result of mechanical obstruction produced by bulging of a hypertrophied interventricular septum into the right ventricle. A number of cases were later described by French and by Latin-American writers (Laubry, 1924; Martini and Joselwick, 1927; Bullrich, 1927 and 1928; Patiro-Mayer and Mazzei, 1928). Until recently, however, the condition has received little attention elsewhere. Fishberg (1937) refers to it in his book on heart failure; Glushein and Geer (1944) report one case; and Russek and Zohman (1945) describe three, of which two were diagnosed during life. The usual cause is hypertrophy of the septum occurring as an integral part of concentric left ventricular hypertrophy in cases of hypertension or aortic valvular disease. The diagnosis can be made clinically where isolated right ventricular failure develops with a lesion that ordinarily causes left ventricular or combined failure.

Rupture of the interventricular septum is an occasional sequel of coronary occlusion. Wood (1944) found thirty-eight reported cases of which eight were diagnosed during life. Rupture generally took place between the third and twelfth days. The onset was associated with substernal pain and shock, followed by appearance of a murmur and thrill. Cyanosis and dyspnœa were present in all reported cases except that of Wood herself, and death commonly resulted from right ventricular failure. Only seven patients lived longer than four weeks, though one survived for four years and ten months.

The present case differs from those in Wood's series in that the rupture of the interventricular septum was incomplete. A dissecting aneurysm developed in the septum itself and produced bulging into the cavity of the right ventricle with obstruction of its outflow tract and signs of right ventricular failure.

Case Report

Miss K., aged 59, had enjoyed good health save for one illness at the age of 49 when she had severe urticaria with vomiting for several weeks. She had no cardiac symptoms prior to April 5, 1946, when she felt sick, vomited, and had pain in the back between the shoulder blades. Pain persisted and she again vomited on April 9, on which day she first summoned Dr. McNab, who found no abnormality of her heart or lungs. She remained in bed for a week and during the next ten days she was up, going about slowly; her pain had subsided and she seemed convalescent. On April 26, she suddenly became breathless but did not send for the doctor until the following day when he noted " extreme pallor, with systolic and diastolic murmurs that had not previously been present." From this time pallor persisted and breathlessness became more severe, but she had no further pain until the day of her death.

She was first seen by me on May 6, 1946. She was of average nutrition with grey complexion, pale cyanosed lips, pale conjunctivæ, and breathlessness. The jugular veins were distended and there was lumbar œdema. The liver was enlarged, extending 4 cm. below the costal margin. The tongue was dry and furred; a faint smell of acetone was noted from the breath. Coarse rales were present over a limited area at the base of each lung. The pulse was of very poor quality, barely perceptible, and it was impossible to obtain a blood pressure reading. The heart rate was 96. A feeble cardiac impulse could be felt, but there was no thrill; the apex was 11 cm. from the midline, and the percussion dullness was increased both to right and to left. The first heart sound was totally obscured by a loud blowing murmur which contrasted sharply with the feeble impulse and barely perceptible pulse; the murmur was heard over a very wide area both in the front and back of the chest; its intensity was maximal along a

* Communication to the III Inter-American Congress, June 1948.

FIG. 1.—Anterior surface of the heart, showing discoloured swollen area produced by the septal dissecting aneurysm. Along the left border just above the apex, an area of slight bulging corresponding to an earlier myocardial infarct can be seen.

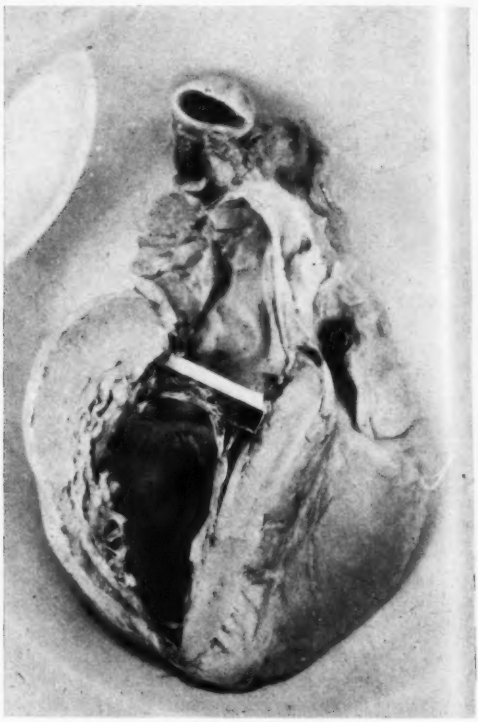

FIG. 2.—Interior of the left ventricle and septum viewed from the left, showing lamellated thrombus on the septum, perforation with everted upper lip, hypertrophy of left ventricle in its basal half, with fibrosis and thinning of the wall in the apical portion.

line joining the fourth left chondro-sternal junction to the apex. The second heart sound was inaudible and no diastolic murmur could be heard.

The patient was admitted to hospital immediately. Following admission she had several bouts of coughing with small hæmoptyses. Her condition deteriorated and she died 33 hours later. A cardiogram showed a Pardee curve of Q I, T I type with a small detached Q I, slightly elevated R–T I, small terminal inverted T I, and slightly negative R–T III. A portable X-ray suggested enlargement of the heart both to left and to right; the superior vena caval shadow was prominent and there was moderate congestion of the lung fields.

Post-mortem Examination (*Dr. J. Adler*). The pericardium contained 35 ml. of straw-coloured fluid.

The heart weighed 460 g. On the anterior surface near the apex was an area of discolouration and swelling (Fig. 1) measuring 55 × 45 mm., corresponding in position to the lower part of the interventricular septum and the apex of the right ventricle. To the left of this, a second area showed slight bulging without discolouration; the myocardium here was thin, clearly representing an old infarct scar with aneurysmal dilatation. Both areas were covered by a thin film of fibrin.

The wall of the left ventricle was hypertrophied, being 23 mm. thick at its base; as it approached the apex it became thin, its thickness being reduced to 1·5 mm. at one point; and here the myocardium was largely replaced by a fibrous scar. The apical half of the interventricular septum was covered by lamellated thrombus; the underlying septal myocardium was necrotic. In the upper and anterior part of the septum there was a round perforation

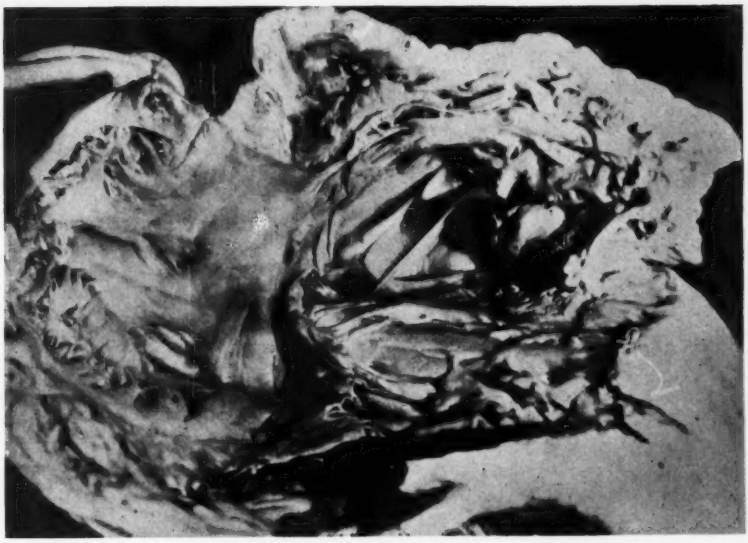

FIG. 3.—Interior of right auricle and ventricle, photographed from the right and below, so as to look into the pulmonary conus across which a glass rod has been placed. The projecting portion of the septal aneurysm is seen on the right of the photograph, at the junction of the septum with the reflected anterior wall of the ventricle. The photograph was made two years after the original examination of the specimen, at which time the aneurysm was opened; considerable shrinkage of the swelling occurred in the interval.

10 mm. in diameter; its upper lip was irregular, ragged, and everted so as to project into the cavity of the ventricle as a small flap (Fig. 2). The perforation led into a dissecting aneurysm of the septum; after running forwards and towards the right till separated from the right ventricular cavity by a layer of tissue only 1 mm. thick, it tracked downwards and forwards towards the apex and anterior wall of the right ventricle. Where it emerged from the septum into the anterior wall of the ventricle, it produced the area of swelling and discolouration seen on the surface (see Fig. 4).

The tissue separating the cavity of the aneurysm from that of the right ventricle consisted of endocardium with a thin layer of intact myocardium. Viewed from the right ventricle, the aneurysm formed an irregularly oval swelling measuring 38 × 28 mm., and projecting into the cavity of the ventricle to a depth of 15 mm. (Fig. 3). This was situated at the junction of the septum with the anterior wall of the ventricle, being so placed as to project into the proximal half of the outflow tract. On opening the aneurysm from the right side it was found to be partly filled by organizing thrombus. The right ventricle was distended, but not much hypertrophied. A small amount of adherent thrombus was present at its apex (Fig. 4).

All valve cusps were healthy. The right coronary artery was calcified along its entire length but was patent. The anterior descending branch of the left coronary artery was the seat of extensive calcification; the vessel was much reduced in calibre throughout its length, and it was occluded by a thrombus at a point 2 cm. from its origin. The circumflex branch of the left coronary artery showed patchy atheromatous calcification.

Calcification was present also in the arteries at the base of the brain. The kidneys showed fibrosis and hyalinization of some glomeruli. The lungs were chronically congested with several recent hæmorrhagic infarcts in each lower lobe. Each pleural cavity contained 200 ml. of fluid. The remaining organs were congested.

DISCUSSION AND SUMMARY

Post-mortem examination showed both old and recent myocardial infarcts. The earlier infarct had

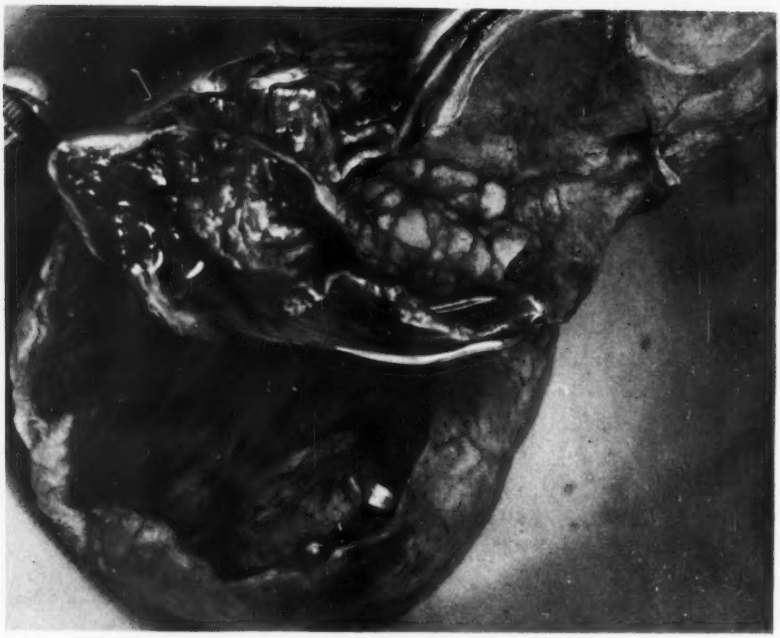

FIG. 4.—Interior of the right ventricle which has been opened along the junction of its anterior wall with the septum; in so doing the aneurysm has been cut through and a portion has been turned back with the anterior wall. The cavity is approximately two-thirds filled with adherent thrombus; its relation to the area of surface discolouration is shown.

affected the left ventricle near the apex producing aneurysmal dilatation of the affected area. This lesion was certainly of some years' standing. Despite the aneurysmal dilatation that followed the initial infarct in a woman who had clearly been hypertensive, there was no admission of incapacity or disability; and the patient's only previous illness did not suggest cardiac disease.

The more recent coronary occlusion gave rise to infarction of the septum and appeared to correspond to the onset of her final illness on April 5, 1946. It was not associated with præcordial or substernal pain but with pain in the back. Perforation into the septum occurred on the twenty-first day; it was associated with sudden breathlessness, pallor, and appearance of a bruit de Roger; there was no thrill. The murmur, which clinically resembled that heard with a congenital ventricular septal defect, is difficult to explain in the absence of a "through and through" perforation. Two possible mechanisms suggest themselves; the everted upper lip of the perforation might have produced such a murmur; alternatively, it may have arisen in the right ventricle from slackening of the chordæ tendineæ and tricuspid curtain, consequent on displacement of the papillary muscle by the aneurysm. The subsequent features of the illness were those of progressive right ventricular failure, coupled with signs suggesting a low cardiac output; they are attributed in part to mechanical obstruction of the outflow tract of the right ventricle by protrusion of the septal aneurysm into it, and in part, to disease of the myocardium. The clinical picture was further complicated by the occurrence of terminal lung infarcts. Death took place on the twelfth day after the rupture.

The fact that rupture in this case led to a dissecting aneurysm instead of a "through and through" perforation, seems to be related to the survival of a thin layer of myocardium immediately beneath the endocardium of the right ventricle; the aneurysm tracked along the line of demarcation between necrotic and intact muscle.

References

Bernheim (1910). *Rev. Médecine*, **30**, 785.

—— (1915). *J. de Practiciens*, Nov. 13, quoted from Glushein and Geer (1944).

Bullrich, R. A. (1927). *Rev. méd. lat.-amer.*, **12**, 31.

—— (1928). *Ibid.*, **13**, 1839.

Fishberg, A. M. (1937). *Heart Failure*, London, p. 432.

Glushein, A. S., and Geer, J. A. (1944). *M. Bull. vet. Admin.*, **20**, 277.

Laubry, C. (1924). *Bull. Mém. Soc. méd. Hôp. Paris*, **48**, 831.

Martini, T., and Joselwick, M. (1927). *Sem. med.*, **34** (11), 11.

Patiro-Mayer, C., and Mazzei, E. S. (1928). *Rev. méd. lat.-amer.*, **13**, 2015.

Russek, H. I., and Zohman, B. L. (1945). *Amer. Heart J.*, **30**, 427.

Wood, Anne M. (1944). *Brit. Heart J.*, **6**, 191.

LONG SURVIVAL WITH A CARDIAC ANEURYSM

BY

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Received July 24, 1948

Although rarely reported, cardiac aneurysms are not purely pathological curiosities that are discovered by chance at autopsy. They can sometimes be recognized during life by correlation of clinical and radiographic findings. Thus, a history of anginal pain, followed by pericarditis and recovery, should suggest the possible development of a parietal aneurysm of the heart, if the patient survives long enough. Further, in spite of the usually accepted view, it is not true to assert that the prognosis of such chronic aneurysms is necessarily bad. Thus, I have been able to follow the development of such a condition for some 13 years in a man who is still free from symptoms.

There are not many reports of long survival after the development of a cardiac aneurysm, but Laubry *et al.* (1930) mention two patients who lived 10 and 12 years respectively, while Clerc and Deschamps (1931) observed survival for 13 years.

My case was that of an agricultural engineer, aged 45, whose illness started with a series of mild anginal attacks, with considerable intervals between them, over a period of about 5 years. On October 25, 1935, after a more severe physical effort than usual, he developed extremely severe præcordial pain, which could not be alleviated completely by any drugs. All the clinical features of coronary occlusion were present—fall of blood pressure, thready pulse, tachycardia, cyanosis, dyspnœa, sweating, anxiety, etc.

Two days later, the temperature rose to 103, accompanied by rigors and signs of left-sided pleurisy. Then, on the fourth day, pericarditis developed, dry at first, but with some effusion later.

All the symptoms diminished in intensity from the twelfth day onwards, the temperature returning to normal, perhaps as the result of giving salicylates. The patient left the hospital 48 days after the original attack.

The radiographic changes are of particular importance, because they have been followed over a period of 13 years.

On the tenth day after the coronary occlusion, the heart was flask-shaped, and no pulsations could be detected, i.e., the appearances were typical of pericardial effusion (Fig. 1).

On the forty-second day of the stay in hospital, the cardiac shadow itself was within normal limits, but there was a small triangular prominence on the left border of the left ventricle, which, at that time, was wrongly interpreted as being due to a pericardial adhesion (Fig. 2). This is now known to have been the early stage of a parietal aneurysm.

Twenty months later, the aneurysm was obvious, the sac being relatively large (Fig. 3). This appearance was detected fortuitously when the chest was being examined because of a suspicion of pulmonary tuberculosis ; but it was not until September 18, 1940, five years after the original attack, that the real nature of the condition was recognized.*

The teleradiograms taken at this time show clearly that the projection is continuous with the left ventricle; and, when screened in various positions, it was seen to dilate during systole of the left ventricle—the condition known as " paradoxical diastole." These appearances, considered in conjunction with the history, demonstrated clearly that the condition was a parietal aneurysm of the heart, not, as had tentatively been supposed, a tumour or a cyst, because these do not pulsate although these may show transmitted pulsation.

Thus, for a period of five years after the attack of coronary occlusion, the diagnosis was uncertain, and had varied from tumour, myocardial cyst, and even a ordinary pericardial adhesion. The present case bears out the old aphorism that, in order to diagnose a disease, it is necessary first to think of it. If we had not considered the possibility of a parietal

* Translator's note. Codounis makes no reference to the possibility that there may have been a pericardial adhesion, which played a part in causing the subsequent aneurysm.

FIG. 1.—31/10/35. Pericardial effusion after cardiac infarction.

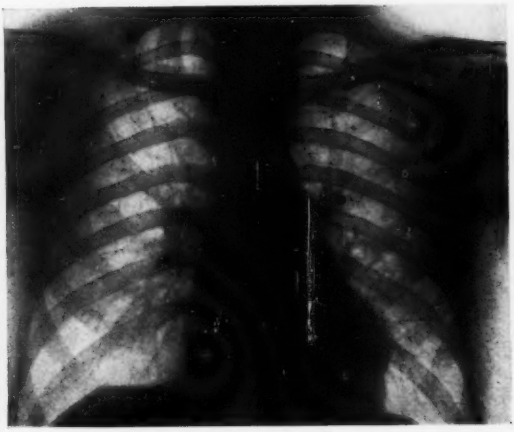

FIG. 2.—3/12/35 (42 days after cardiac infarction). A small projection is seen on the left lateral wall of the left ventricle.

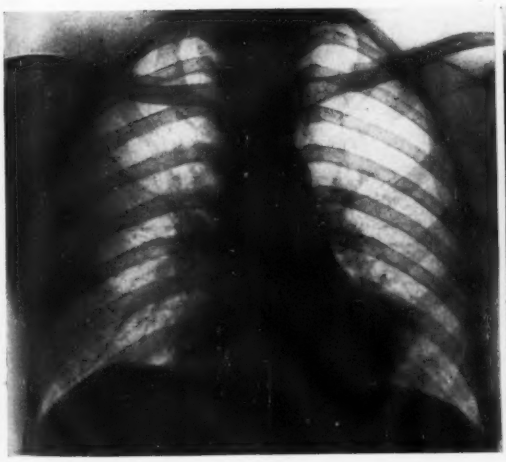

FIG. 3.—3/8/37. A large aneurysmal sac is visible.

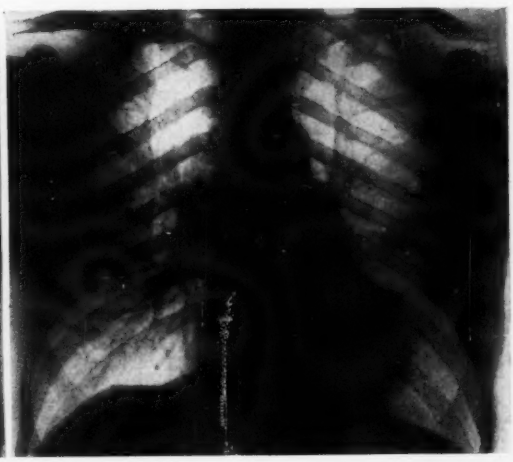

FIG. 4.—18/7/40. The sac is still present, but shows a rather diffuse edge.

aneurysm, we should not have attempted to demonstrate it by looking for paradoxical diastole: a further proof, if one be needed, of the value of screening in cardiology.

Fig. 4, which was taken seven years after the attack of pericarditis, shows slight decrease in the size of the sac, which still shows the phenomenon of paradoxical diastole, although its density has changed a little, now being different from that of the ventricle. Oddly enough, paradoxical diastole was even more striking than at earlier examinations.

Towards the end of 1945, fifteen years after the beginning of the illness, the patient, who felt perfectly well, showed no clinical signs of heart disease except occasional slight anginal pain on exertion or after repeated bending. From this time onwards, there has been little change in the radiographic appearances, except that the sac shows areas of increased density, which are, presumably, due to calcification: a feature which was more easily recognized on screening than in films. In spite of this, pulsation of the aneurysm was still distinctly visible.

In our view orthodiagraphy is of less value than teleradiography and kymography, mainly because of the subjective element in the former, which leads to considerable differences between the orthodiagrams made by different radiologists, even on the same day.

It seems clear that the radiographic history of the cardiac aneurysm recorded here has all the features that would be expected from our knowledge of myocardial infarction. And the fact that the aneurysmal sac plays only a passive part during ventricular systole is consistent with the replacement of muscle fibres by scar-tissue.

Differential diagnosis is not difficult in cases in which the phenomenon of paradoxical dilatation of the aneurysmal sac can be detected; but, if the contents become thrombosed or the wall becomes calcified, this may disappear. Then, the possibility of such conditions as hydatid cyst, neoplasm, pericardial diverticulum, etc., will need to be considered.

Again, large aneurysms of the heart may cause such great alteration in the cardiac outline as to cause possible confusion with pericardial effusion, but the invisibility of the heart-beat in that condition should prevent mistakes.

Finally, coronary aneurysms present an identical radiographic appearance, but usually occur on the right, whereas parietal aneurysms are almost invariably left-sided. Then also, coronary aneurysms are due to syphilis and develop insidiously, without any history of the type found in coronary thrombosis.

Summary

A case illustrating the development of an aneurysm of the heart-wall during a period of 13 years is recorded in detail; and special attention is drawn to the occurrence of paradoxical diastole as an important diagnostic feature.

References

Clerc, M. A., Deschamps, N. (1931). *Cœur et vaisseux in Précis de Pathologie Médicale*, Masson, Paris.

Laubry, C. *et al.* (1930). *Maladies du cœur et des vaisseaux*, Doin, Paris.

U WAVE INVERSION

BY

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Received July 7, 1948

The U wave, when first recognized by Einthoven (1906) in the earliest days of electrocardiography, was considered by him to be pathological. Since then the normality of upright U waves, except perhaps in cases where they are unusually high (Katz, 1946), has been generally conceded. Various hypotheses have been advanced to explain the U wave, of which the most satisfactory is that of Hoff and Nahum (1938) who concluded on experimental grounds that it undoubtedly forms part of the ventricular complex, and is coincident in time with the supernormal phase.

The number of papers on the clinical significance of the U wave is small, and reflects the scant attention that seems to have been paid to it by most cardiologists. Important articles however are those of Nahum and Hoff (1939), Papp (1940), and Solarz and Elek (1943), and to them the reader is referred for comprehensive reviews of the subject.

Inversion of U has been considered pathological by most authors. Nahum and Hoff found it in association with coronary, rheumatic, hypertensive, and pulmonary heart disease. Papp reported it in coronary disease and in hypertension. Solarz and Elek found 94 cases of U wave inversion in 1000 cases studied; they report on its association with the left heart strain pattern, intraventricular block, and anterior and posterior wall infarction.

Negative U in Induced Angina

The writer's interest in negative U waves was stimulated by noticing their appearance in the after-exercise tracing of a patient with angina pectoris. Subsequently six further such patients were found. All were males, and in all of them pain was easily induced by effort. None gave a history of a previous clinical attack of coronary occlusion. Six of the seven had hypertension; all had cardiac X-ray silhouettes that were within the normal range. In all except one (Case 7) test exercise was given to the point of just inducing pain, and the tracing was made as soon afterwards as possible. Table I shows the degrees of U wave positivity and negativity both before and after exercise; in Table II are listed any other abnormal features of the electrocardiograms, as well as certain clinical data.

TABLE I

THE U WAVE BEFORE AND AFTER EXERCISE IN ANGINA PECTORIS

Case		Lead I	Lead II	Lead III	Lead CF 2	Lead CF 4	Lead CF 5
1	Before	0	0	+	+	−	−
	After	−−	+	++	−−	−−−−	−−−−
2	Before	0	+	+		+	
	After	*	+	+		−−	
3	Before	+	+	0	+	+	+
	After	−	+	+	−	−−	−−
4	Before	0	0	+	++	+	−−
	After	−	0	+	+	−−	−−
5	Before	−	+	+	−	−−	−
	After	±	+	+	−−−	−−−−	−
6	Before	0	−	−	0	−−	0
	After	*	*	*	−	−−	−
7	Before	0	−−	−	++	+	0
	After **	±	−−	−−	++	+	0

0 = isoelectric. + or − = less than 0·5 mm.
+ = positive. ++++ or −−−− = 1·5 to 2·0 mm.
− = negative. ± = diphasic.
*Pulse rate 100 or more.
**Exercise not carried to point of inducing pain.

In the control (or before-exercise) tracings, U was negative in one or more leads in five of the seven cases. In two of this five U was inverted in the chest leads only, in two it was inverted in both chest and limb leads, and in one in limb leads only.

It will be seen from Table I that in the six cases in which exercise was carried to the point of inducing pain, U became, after exercise, either: (a) more negative if it was already inverted; (b) negative in additional leads in which it had previously been positive or isoelectric; or (c) both (a) and (b). Maximum negativity in these six was always in one

TABLE II

FINDINGS, IN SEVEN PATIENTS WITH ANGINA PECTORIS SHOWING U WAVE INVERSION

Case	Age	B.P.	Duration of Angina	Electrocardiogram	
				Before Exercise	*After Exercise*
1	46	250/130	2 mth.	Intraventricular conduction defect (QRS=0·11 sec.); T negative in I, CF 2, CF 4, and CF 5.	Depression RS–T in I; less negativity T in chest leads.
2	65	190/95	1 yr.	Diphasic T in CF 4.	Depressed RS–T in II and III; positive T in CF 4.
3	43	160/105	2 yr.	None.	Depression RS–T in CF 4; "coronary" type positive T waves in CF 4 which later became negative.
4	61	170/190	10 yr.	Diphasic T I and T II; negative T in CF 5.	Depression RS–T in CF 4; "coronary" type positive T waves in CF 2 and CF 4.
5	36	120/105	3 mth.	Negative T in CF 2, CF 4, and CF 5.	T positive and of "coronary" type in CF 2 and CF 4.
6	43	120/80	3 mth.	Notching of S in CF 2; depressed RS–T (0·5 mm.) in CF 4.	Depression RS–T in CF 4.
7	58	190/90	5 yr.	None.	None.*

* Exercise not carried to point of inducing pain.

or more of the chest leads. In five of them it was possible to recognize a pattern consisting of inversion in lead I and in the chest leads, greatest in CF 4 with positivity of U in lead III (see Fig. 1); in one case a positive U appeared in lead III where previously it had been isoelectric, and in another the positivity of U in lead III increased. This pattern is reminiscent of the pattern of T wave inversion seen in anterior coronary occlusion. In another case U was negative in leads II, III, and CF 4 before exercise, and after exercise became negative also in CF 2 and CF 5. In Case 7, where exercise was not carried to the point of inducing pain, U was negative in II and III, but positive in CF 2 and CF 4.

In three cases there developed with exercise, in certain of the chest leads, an interesting pattern comprised of sharply pointed positive T waves with symmetrical shoulders which have previously been described as "coronary T waves" (Katz, 1946), and negative U waves (see Fig. 2 and 3). This pattern has been seen by us only in patients with coronary insufficiency; whether or not it should be considered pathognomonic of this condition will depend upon further observations.

A discordancy in the direction of the development of T wave and U wave potentials was seen in two patients. In Case 5 (see Fig. 3) the T wave had, in the control tracing, been negative; its change to positive with exercise was concurrent with the development of increased negativity of U. Conversely, in Case 3 (see Fig. 2) a negative U, which made its appearance immediately after exercise, was found to disappear five minutes later at the same time as T became negative.

NEGATIVE U AS ISOLATED ABNORMALITY

Tracings in which U wave inversion constitutes the only cardiographic abnormality are probably uncommon. Nahum and Hoff (1939) reported two cases, but the high T wave in lead IVR in one of them (their Fig. 8) is probably abnormal. In Papp's (1940) case there is a pathologically low takeoff of the RS–T segment in IVR. Solarz and Elek (1943) state that "no abnormal U waves were found in the absence of other abnormalities in the electrocardiogram," and that therefore "there is not much diagnostic value in the recognition of U wave patterns." It seems difficult however for them to justify this conclusion, in view of their having studied only tracings that had been chosen for their abnormality. Katz (1946) observes that abnormalities of U rarely occur in the absence of abnormalities of S–T–T and believes that "little clinical weight would be given to an electrocardiogram in which the only deviation from the normal was in the U wave."

The writer has records of three cases in which no trace of abnormality other than U wave inversion could be made out. The first patient (see Fig. 4) suffered from Paget's disease and severe hypertension, and at the time that the tracing was made was actually in congestive failure. The second had angina pectoris (Case 7 of present series). The third was a patient suffering from polycystic kidney disease with uræmia and severe hypertension.

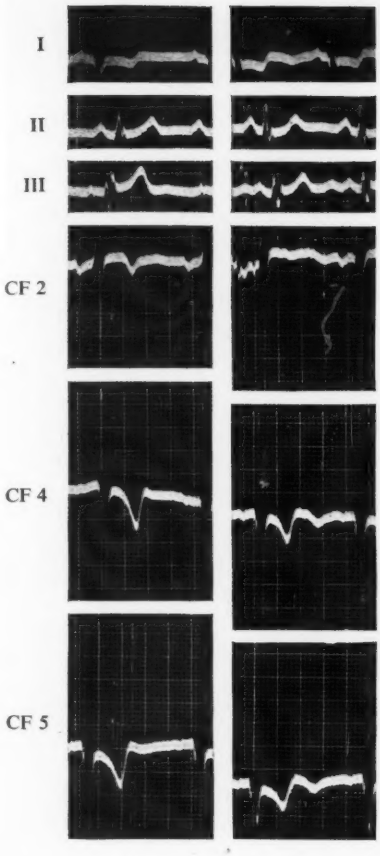

FIG. 1—Case 1. Man, aged 46, with severe hypertension and angina pectoris. Tracings (A) before and (B) immediately after pain-inducing exercise. Note wide QRS. With exercise negative U appears in leads I and CF 2; U becomes more negative in CF 4 and CF 5, and more positive in III. Fig. reduced by ⅓.

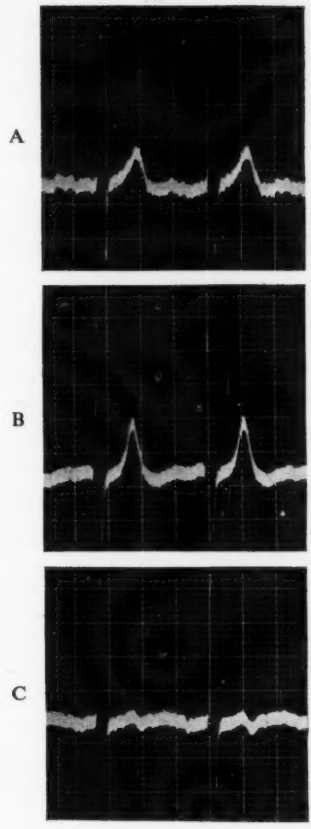

FIG. 2—Case 3. Lead CF 4. (A) Before, (B) immediately after, and (C) five minutes after. Note appearance of "coronary T" and negative U immediately after exercise. Five minutes later T has become negative and U isoelectric.

In another patient with mitral stenosis the only additional abnormalities were a large P wave in lead II and a prominent S in lead I; the U wave was only detected when the tracing was compared with a previous one taken when the patient had been digitalized (see Fig. 5).

In none of the tracings made immediately after pain-evoking exercise was inversion of U the sole abnormality. In two cases however the only other findings were a slight depression of the RS–T segment in lead CF 4, and the appearance of symmetrical T waves.

NEGATIVE U IN INTRAVENTRICULAR BLOCK

U wave inversion in bundle branch block is described by Nahum and Hoff (1939) and by Papp (1940). Solarz and Elek (1943) failed to find it in 75 consecutive cases of intraventricular block of the S type, but saw it in 17 of 135 cases of the common type: the general pattern was negativity in leads I

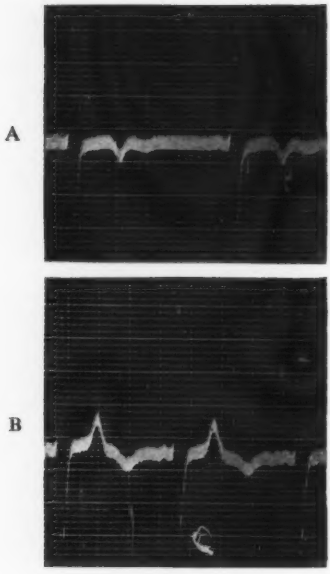

FIG. 3—Case 5. Lead CF 4. (A) Before and (B) after exercise. Note development with exercise of positive "coronary T" concurrently with increase in negativity of U.

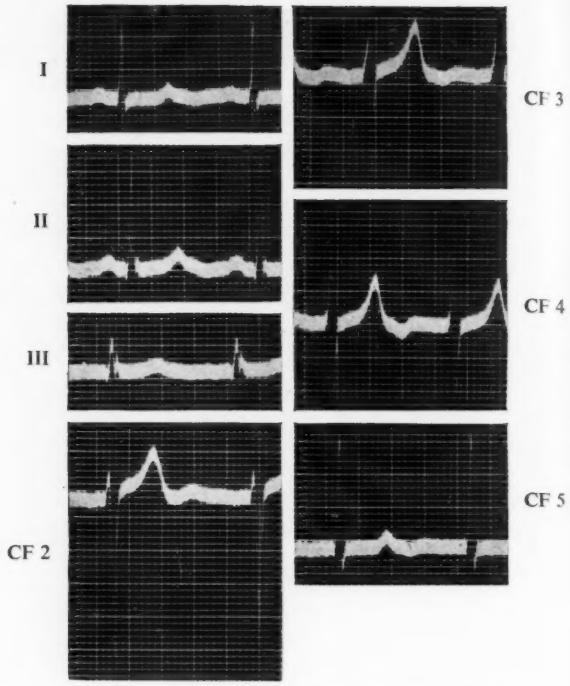

FIG. 4—Case 8. See text. Note U wave inversion as only abnormality: negative U in I and II and in the left chest leads with diphasic transition U wave in CF 3.

and CF 4, with upright or isoelectric U waves in leads II and III.

In Case 1 of the present series QRS had a duration of 0·11 sec. and its configuration was of indeterminate type. The patient's pain was easily induced, and appeared when the heart rate had risen from 62 to only 75; the sole significant resulting cardiographic changes were pronounced alterations in the U wave (see legend, Fig. 1). It is difficult not to conclude that the U wave changes here, and perhaps also those found in other cases where there was an associated intraventricular block, are actually related to myocardial alterations rather than to conduction defects as such.

VARIATION OF R–U DURATION

We have not been able to corroborate Papp's (1940) observation that with exercise the R–U time remains relatively constant irrespective of the heart rate. On the other hand we found in two of our cases that R–U was shortened by as much as 0·12 sec. during exercise, even though the heart rate increased only from 62 to 80, and from 65 to 85 respectively.

SUMMARY AND CONCLUSIONS

The appearance of temporary U wave inversion during anginal attacks induced by exercise is described.

The pattern of U wave inversion seen most frequently is made up of negativity in lead I and in the left chest leads, and positivity in lead III.

The association in individual chest leads of sharply pointed, symmetrical, positive T waves with inverted U waves in patients with angina pectoris during exercise is described.

Discordancy in direction of development of T and U may occur in the chest leads during exercise.

Four cases are described of inversion of U in one or more leads as an isolated electrocardiographic abnormality.

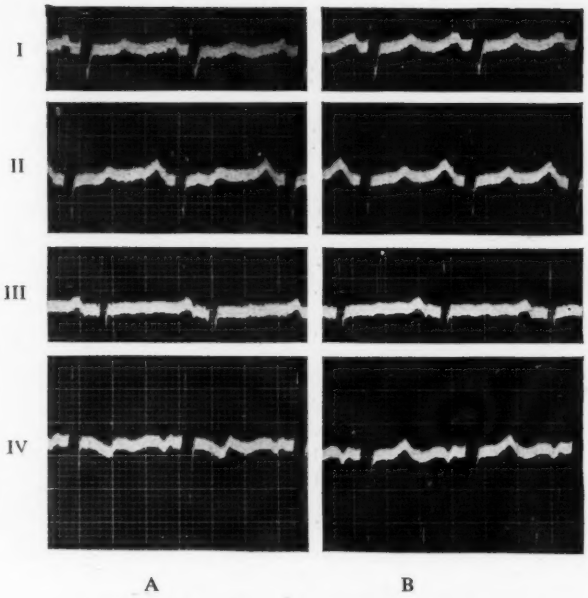

FIG. 5—Case 9. Woman, aged 39, with mitral stenosis. Tracings taken (A) during digitalis therapy and (B) ten days after cessation. Note that T IV has become positive but that negative U waves in I and IV have persisted.

Inversion of U when seen in tracings showing bundle branch lesions is probably the result of associated myocardial damage rather than of the conduction defect as such.

The R–U time interval may decrease as the heart rate increases.

Inversion of U in one or more leads should always be considered pathological.

REFERENCES

Einthoven, W. (1906). *Arch. internat. Physiol.*, **4**, 132.
Hoff, H. E., and Nahum, L. H. (1938). *Amer. J. Physiol.*, **124**, 591.
Katz, L. (1946). *Electrocardiography*, 2nd ed., Philadelphia, p. 121.
Nahum, L. H., and Hoff, H. E. (1939). *Amer. Heart J.*, **17**, 585.
Papp, C. (1940). *Brit. Heart J.*, **2**, 9.
Solarz, S. D., and Elek, S. R. (1943). *J. Lab. clin. Med.*, **28**, 936.

CARDIAC LESIONS IN THIAMIN DEFICIENCY

BY

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Received July 22, 1948

Experience of beriberi occurring among British and Australian prisoners of war indicated that heart block was not infrequent.

There is no general agreement about the effect of thiamin deficiency on the heart, and in particular on cardiac conduction. The only electrocardiographic abnormality noted by Aalsmeer and Wenckebach (1929) was a shortening of the P–R interval. Keefer (1930) in 29 cases of beriberi noted no characteristic cardiographic change, though cardiac conduction was sometimes slightly altered: 15 of the 29 cases had evidence of cardiac insufficiency; these showed enlargement of the right auricle, right ventricle, pulmonary conus, and pulmonary artery. Jolliffe (1939) in experimental thiamin deficiency found cardiographic changes in 2 of 5 human volunteers: in one, sinus arrhythmia, sinus arrest, and inversion of T III appeared after 11 days of deficiency, and in a second inversion of T III appeared after 8 days of deficiency. The changes reported by Weiss and Wilkins (1937) and by Dustin *et al.* (1939) included tachycardia, prolongation of electric systole, low voltage complexes, and flattening or inversion of T waves. Dock (1940) recorded 5 cases of idiopathic cardiac hypertrophy with mural thrombi thought to result from thiamin deficiency, in 4 of whom there was disturbance of conduction—right bundle branch block in 3 and latent A-V block in 1 case. Casanova (1946) observed flat bifid P waves, prolongation of the P–R interval, and auricular fibrillation (once) in beriberi.

Most accounts of the pathology of the heart in beriberi have been limited to a description of the macroscopic abnormalities. It is uncertain whether the enlargement of the right side of the heart is due to dilatation alone or to dilatation combined with hypertrophy.

Weiss and Wilkins (1937) found that the weight of the heart was generally normal and that there was a moderate dilatation of the right ventricle. Wenckebach (1934) and Weiss and Wilkins (1937) observed a " hydropic " degeneration of the muscle fibres.

Disturbances of A-V conduction are not mentioned in most clinical accounts of beriberi and lesions affecting the specialized muscle of the conducting system have not been noted on histological examination. Thus Blankenhorn (1945) gave as the first of the criteria for the diagnosis of beriberi heart disease " an enlarged heart with normal sino-auricular rhythm."

As there is no general recognition of the appearance of conduction defects in human thiamin deficiency it was thought advisable to record the clinical impression of its not infrequent occurrence. In the light of our clinical experience it was considered that analysis of the effects upon cardiac conduction of prolonged severe thiamin deficiency in animals would be of value and should determine whether the changes present had a functional or organic basis.

Experimental Methods

Pigs were used since they are known to be the animals most susceptible to thiamin deficiency. The animals were obtained when five weeks old. The dietetic method was largely that of Wintrobe (1942). This consists of feeding a basal vitamin-free diet. The basal diet in this experiment consisted of sugar, crude casein and cooking fat, and provided approximately 150 calories per kg. of body weight daily. The daily supplements added to this diet were those used by Wintrobe (1943). With the exception of thiamin which was entirely omitted, these supplements contained synthetic vitamins and minerals in quantities known to be adequate to maintain the normal health and growth of the animals.

Four pigs were kept on this diet. Two additional pigs used as controls were given the same basal vitamin and mineral supplement, and in addition these control pigs were given 0·51 mg. of thiamin hydrochloride per kg. of body weight daily.

Frequent electrocardiograms were obtained from all six animals. Three standard leads and one chest lead CF 4 were used. The normal variations in the

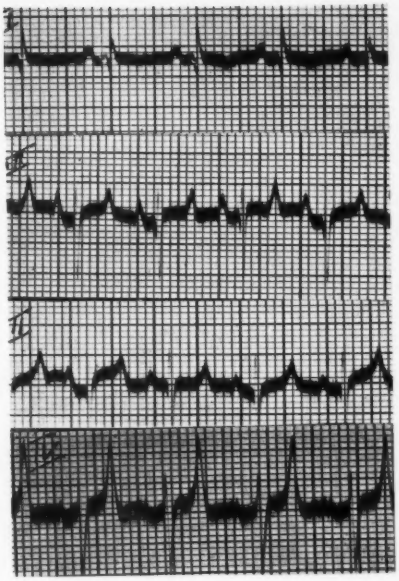

FIG. 1.—Normal pig. Standard limb leads and CF 4. The T wave in CF 4 is invariably upright.

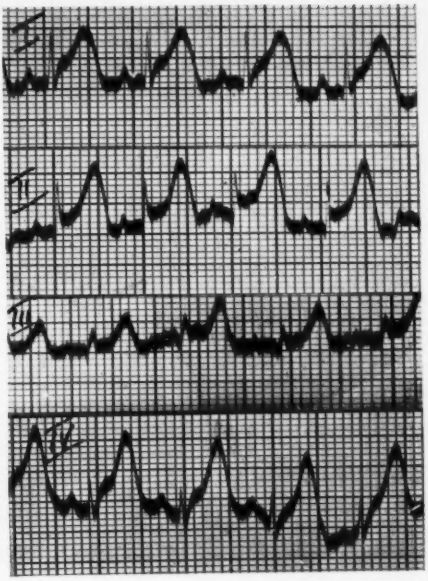

FIG. 2.—Pig No. 1. Standard leads and CF 4 on 73rd day of deficiency, 24 hours before death. Huge T waves and elevation of S–T segment in all leads.

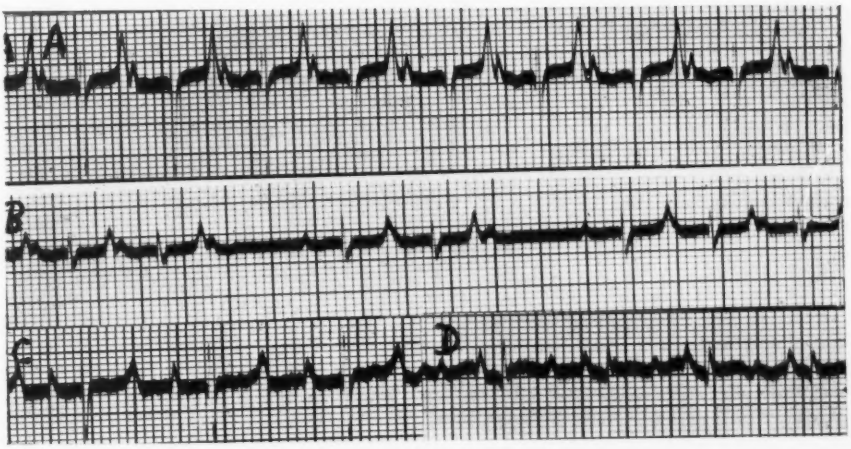

FIG. 3.—Pig No. 1. (A) Lead II: 40th day of deficiency, showing first degree of A-V block. P–R interval 0·18 sec. (B) Lead I, recorded 4 hours later showing second degree A-V block. (C) Lead II: 41st day of deficiency, 24 hours after injection of 2·5 mg. thiamin. P–R interval 0·16 sec. (D) Lead II: 45th day of deficiency. P–R interval has returned to within normal limits.

pig were established at the beginning of the experiment. The average heart rate was 143, and the lowest rate recorded was 130. The rhythm in the normal pig was invariably regular, and in no instance was sinus arrhythmia recorded. The P–R interval varied from 0·06 to 0·10 sec. usually 0·08 to 0·10. The QRS varied from 0·04 to 0·08 sec. T I was frequently inverted and T II and III were usually upright. Inversion of T in CF 4 was not seen in the normal pig (Fig. 1).

Results

Control pigs maintained on this regime with adequate daily thiamin allowance remained healthy.

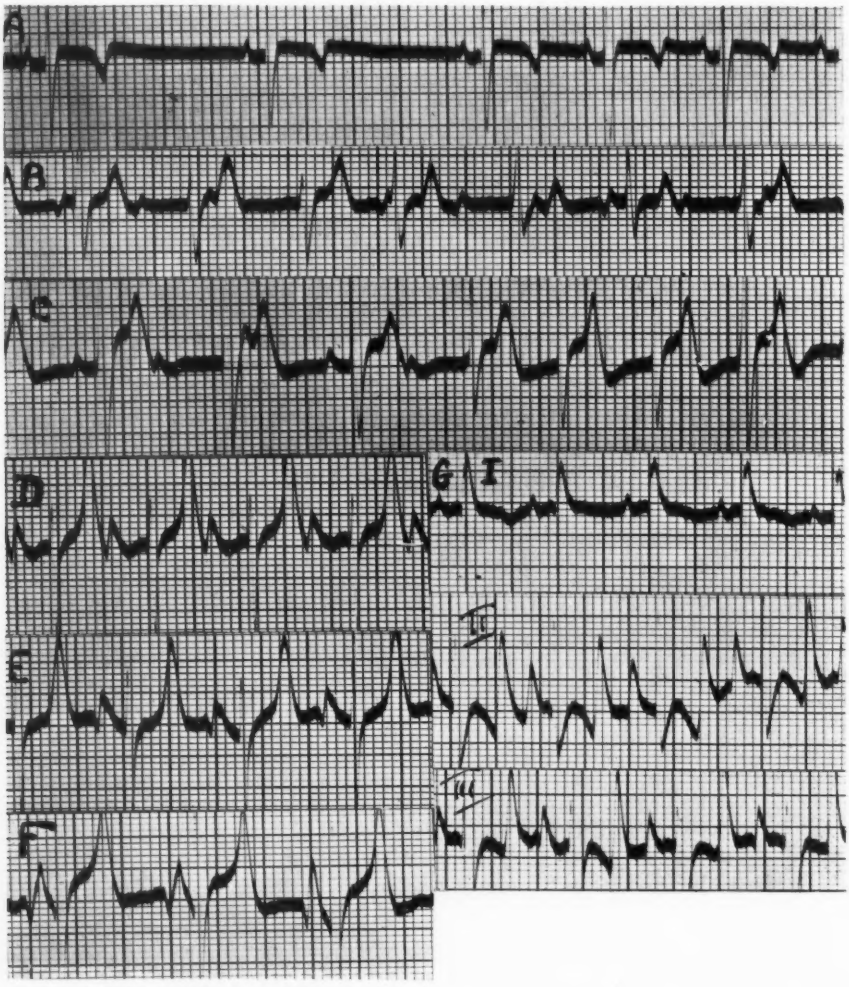

Fig. 4.—Pig No. 2. (A) Lead IV: 49th day of deficiency showing sinus arrest and inversion of T IV. (B) and (C) Leads I and IV: 77th day of deficiency showing complete heart block and elevation of S–T segment in lead IV. (D) Lead II: 77th day, one hour after injection of 2·5 mg. of B_1. First degree block, P–R interval 0·18 sec. (E) Lead II: 78th day, 24 hours after thiamin, P–R interval 0·16 sec. (F) 80th day P–R interval within normal limits. (G) Standard leads: 99th day; left bundle branch block, the QRS interval exceeds 0·08 sec.

Frequent electrocardiograms showed no deviation from the normal. These control pigs were slaughtered on the 84th experimental day and their hearts subjected to detailed histological examination. Numerous sections from all four chambers of the heart, and multiple sections from the interauricular and interventricular septa failed to show any histological abnormality.

The four pigs deprived of thiamin developed anorexia and occasional vomiting between the 30th and 40th days, and thereafter at intervals. Three of the animals died from thiamin deficiency. One was sacrificed after 157 days. All four developed disturbances of A-V conduction (Fig. 3, 4, 5, and 6). The animals were acutely ill at the time of appearance of A-V block. One animal (No. 3) died with complete block.

Death of the others showing block was prevented by a single intramuscular injection of 2·5 mg. of thiamin hydrochloride. The time of appearance of the conduction disturbance, the degree of block, the response of the block to thiamin, and the duration of survival on the thiamin deficient regime are indicated in the table. From this it is seen that the sinus arrest, and inversion of CF 4 (Fig. 4, 5, and 6). These changes appeared between the 40th and 60th days. Sinus block was not affected by atropine.

Normal cardiograms were recorded for some time following the treatment of A-V block with thiamin. Pig No. 2 developed left bundle branch block 22 days after thiamin treatment of complete block. The other two animals failed to develop a second episode of block but before death showed marked elevation of the S–T segments and huge T waves on all four leads (Fig. 2).

Post-mortem examination of these animals showed no evidence of subcutaneous œdema. Excess of fluid was found in the pericardial sacs. In one animal (No. 4) this amounted to 170 ml. (6 oz.). No excess of fluid was apparent in the other body cavities. The hearts showed dilatation of the right auricle and of the right ventricle, particularly in the region of the pulmonary conus. Apart from the heart of animal No. 2 which showed numerous subendocardial hæmorrhages, considered incidental to the shock of killing, there was no macroscopic abnormality in the muscle, valves or endocardium. The hearts formed 0·51 to 0·59 per cent of the body weight as opposed to the normal 0·3 to 0·4 per cent.

TABLE

DEVELOPMENT OF HEART BLOCK WITH THIAMIN DEFICIENCY

Animal	Day of deficiency at the appearance of block	Degree of block	P–R interval: (*a*) 1 hr. (*b*) 24 hrs. (*c*) 48 hrs. after injection of thiamin	Day of death
No. 1..	40th (1) Morning. (2) Afternoon	Latent P–R, 0·2 sec. 2nd degree	(*b*) 0·16 sec. (*c*) 0·1 sec.	74th
No. 2..	77th 99th	Complete Left bundle branch	(*a*) 0·18 sec. (*b*) 0·16 sec. (*c*) 0·1 sec.	Sacrificed 157th
No. 3..	63rd 64th	2nd degree Complete		64th
No. 4..	58th to 63rd	Latent P–R, 0·14–0·15 sec.	(*b*) 0·08 sec.	116th

administration of thiamin to three of the animals during the episode of block resulted in return of normal A-V induction within 48 hours. Following thiamin treatment the animals were maintained on the deficient diet.

With the exception of pig No. 1 in which heart block occurred on the 40th day, the animals showed cardiographic changes prior to the development of block. These consisted of marked bradycardia—rates as low as 65 were recorded—sinus arrhythmia,

Sections for histological examination were obtained from the anterior and posterior walls of each auricle, from the auricular appendages, the interauricular septa and from the anterior, lateral, and posterior walls of each ventricle. The interventricular septa were blocked in series and multiple sections from each examined.

Lesions were found in the auricles, the auricular

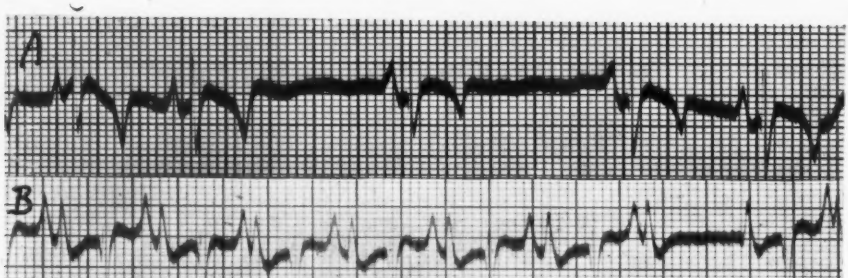

FIG. 5.—Pig No. 3. (A) Lead IV: 52nd day of deficiency, showing sinus block and inversion of T IV. (B) Lead II: 63rd day of deficiency, showing second degree A-V block. (C) Lead I: 65th day, half-an-hour before death, showing complete heart block.

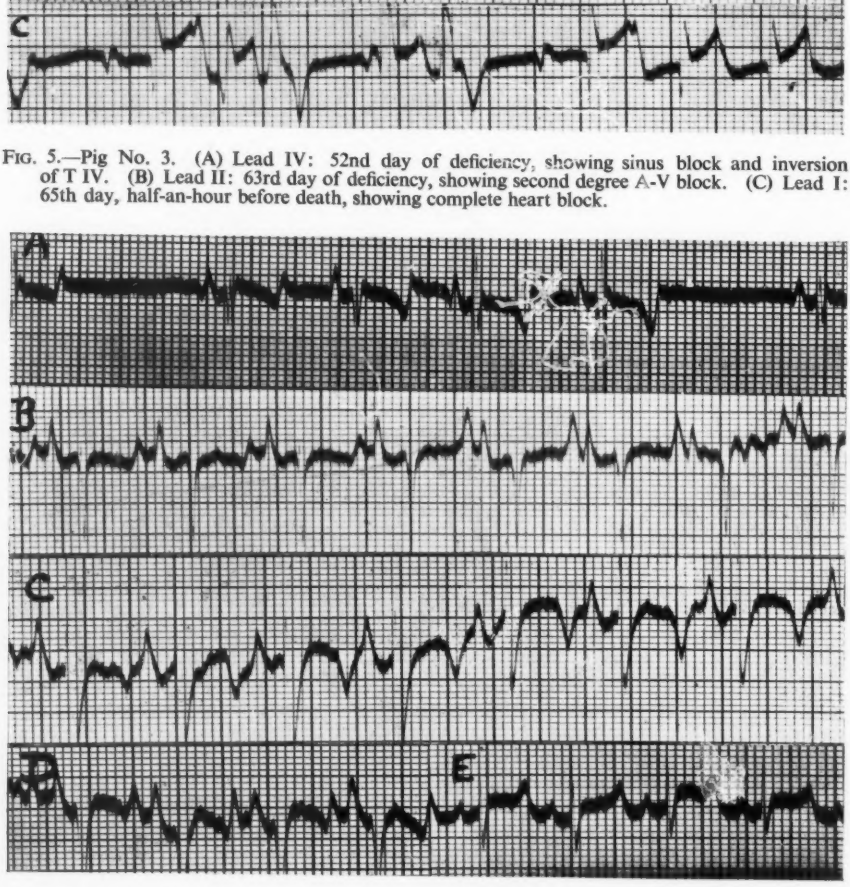

FIG. 6.—Pig No. 4. (A) Lead II: 52nd day of deficiency, showing sinus arrest. (B) and (C) Leads II and IV: 62nd day of deficiency, showing first degree A-V block (P–R 0·16 sec.) and inversion of T IV. (D) and (E) leads II and IV, following the administration of thiamin. The P–R interval is within normal limits. T IV is upright.

appendages and the auricular septa in all four animals. These consisted of areas where muscle fibres had disappeared leaving an œdematous reticulum the interstices of which frequently contained an eosinophilic and protein-like fluid. A mononuclear cellular infiltration was seen in the areas of muscle damage and in the interstitial tissue in relation to them. Changes of a more acute character appeared in the left auricle and auricular septum of animal No. 4. Here were seen areas of active muscle necrosis associated with a leucocytic exudate and gross extravasation of red cells (Fig. 7).

Healing and healed lesions in the auricles of three of the animals were minimal. However in pig No. 2 which survived 157 days, areas of replacement of muscle by granulation tissue or fibrous tissue were numerous. Some sections from the left auricle of this animal showed replacement of the majority of the muscle fibres by fibrous tissue (Fig. 8).

The microscopic auricular lesions old and recent, were in all the animals most marked in the left auricle and auricular septum. In sections taken from the ventricular septum and ventricular walls it was possible to demonstrate the bundles of His, its main branches, and the terminal subendocardial and intra-myocardial ramifications of the conducting system.

The conducting system in the pig differs markedly from that in man. Glomset and Glomset (1940) have shown that in man Purkinje cells do not exist in the main bundle nor in the upper part of the right branch. The cells that are described as Purkinje elements in the left branch and lower part of the right branch differ markedly from those found in ungulates. They do not form a network, are found only subendocardially, and have never been recognized within the myocardium. Cells closely resembling those described by Purkinje in the heart of the sheep are seen in the bundle of His, and its main branches in the pig. Similar cells are seen forming a subendocardial network in the ventricles and penetrating deeply into the ventricular myocardium. These cells are round, oval, or band-like in shape with distinct cell outline and measuring from 24μ to 66μ in diameter. The cytoplasm is eosinophilic staining irregularly and shows a perinuclear clear zone. The myofibrils of the cells are irregular in their arrangement. The cells occasionally show a double nucleus; more commonly they have a single elongated nucleus which varies in its position in the cell. In elongated cells the long axis of the nucleus tends to be arranged at right angles to the long axis of the cell.

The ventricular lesions in these animals were confined almost entirely to the Purkinje system. In animal No. 3 which had at the time of death complete heart block, marked recent degenerative changes were found in many of the cells of the conducting system. These degenerate cells had shrunken from their surrounding supporting reticulum, their

FIG. 7.—Pig No. 4. Interauricular septum. There is an acute hæmorrhagic necrosis of muscle fibres. Magnification × 210.

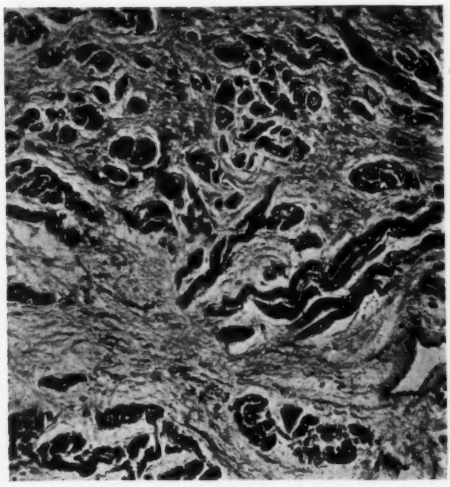

FIG. 8.—Pig No. 2. Section from the right auricle showing the healed auricular lesions. There is marked fibrous replacement of muscle. Magnification × 90.

FIG. 9.—Pig No. 3. Subendocardial Purkinje cells. (A) Normal cells. The myofibrils are distinct. (B) Necrotic cell. The cell stains deeply. The myofibrils are no longer apparent. Magnification × 375.

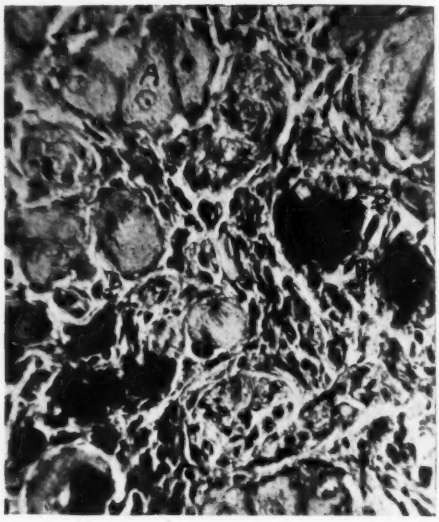

FIG. 10.—Pig No. 3. Bundle of His from the animal dying with complete heart block. There is an acute hyaline necrosis of many of the Purkinje cells. (A) Normal cells. (B) Necrotic cells. Magnification × 375.

cytoplasm had become deeply eosinophilic and of a structureless hyaline appearance. The myofibrils were no longer distinguished (Fig. 9). The nuclei showed pyknotic change.

More than half the cells in the main bundle (Fig. 10) and many of the cells in the main bundle branches were involved in this acute hyaline necrosis (Fig. 11). One-third to one-half of the subendocardial and intra-myocardial Purkinje cells were affected. A mononuclear cellular reaction was found surrounding many of these degenerate subendocardial and intra-myocardial cells. Cellular reaction was much less evident in relation to the damaged cells in the bundle of His and its main branches.

Sections from the main bundles of pigs No. 1 and 4—these animals had survived episodes of heart block—showed in comparison with sections from the bundles of normal animals a complete disappearance of a considerable number of Purkinje cells and their replacement by connective tissue proliferation (Fig. 12, 13, and 14). Many of the remaining Purkinje cells showed an acute hyaline necrosis. These changes, disappearance of Purkinje cells and hyaline necrosis in a proportion of the remaining cells were also evident in the bundle branches (Fig. 15) where in addition a mononuclear cellular reaction surrounded some of the damaged cells. The terminal subendocardial and intramyocardial ramifications of the Purkinje system throughout the ventricles of these two animals were grossly involved in both old and recent lesions. More than half the Purkinje cells showed recent necrosis with an associated marked mononuclear cellular reaction. In some sections complete interruption of a Purkinje strand by a cellular exudate was seen (Fig. 16).

Acute hyaline necrosis of the Purkinje cells was not found in the animal sacrificed after 157 days. Extensive healed lesions consisting of fibrous replacement of Purkinje cells were however found throughout the conducting system, and were particularly marked in the bundle branches.

DISCUSSION

Records of the cardiovascular changes in experimental thiamin deficiency in animals are numerous. In thiamin deficient rats, Weiss *et al.* (1938) found marked bradycardia and changes in the T waves and S–T segments; these changes usually disappeared within a few hours of giving thiamin, but were unaffected by atropine. Carter and Drury (1929) noted bradycardia and heart block in thiamin deficient pigeons. Swank and Bessey (1942) however, minimize the significance of bradycardia and heart block in pigeons since they found starvation

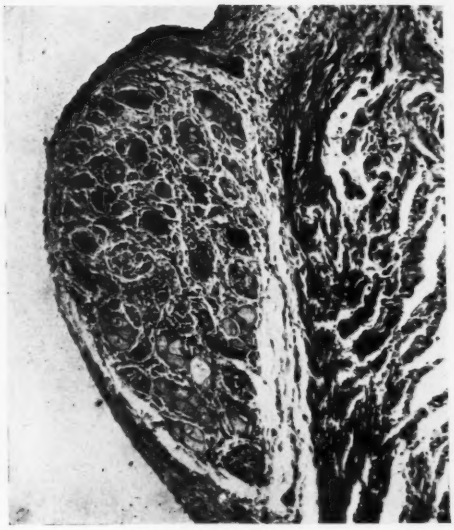

FIG. 11.—Pig No. 3. Left bundle branch showing acute hyaline necrosis of approximately one-third of the Purkinje cells with associated cellular reaction. Magnification × 75.

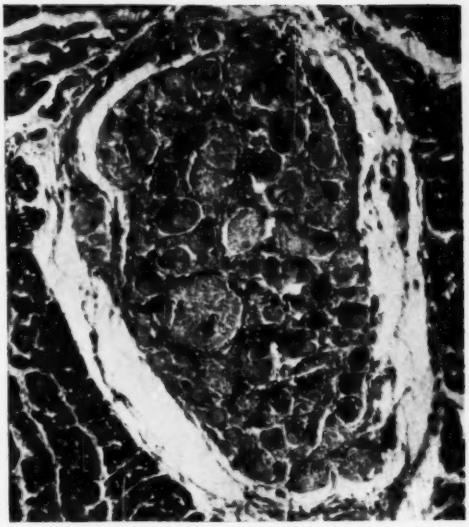

FIG. 12.—Bundle of His in a normal pig. Magnification × 60.

FIG. 13.—Pig No. 1. Bundle of His from an animal surviving 34 days after the occurrence of heart block, showing loss of Purkinje cells and their replacement by connective tissue. A number of the remaining cells show a hyaline necrosis. Magnification × 60.

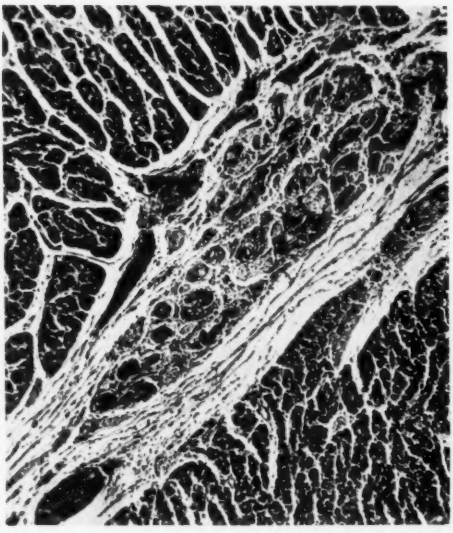

FIG. 14.—Pig No. 4. Bundle of His from an animal surviving 53 days after the occurrence of block. The changes are more marked than those in Fig. 13. Magnification × 40.

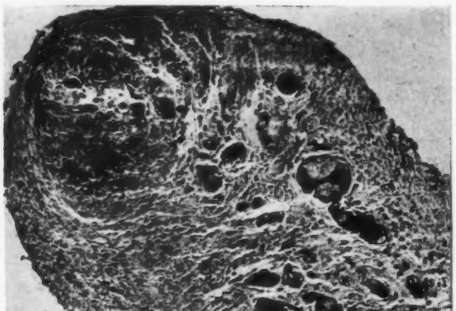

FIG. 15.—Pig No. 1. Terminal part of the left bundle branch showing loss of Purkinje cells and fibrosis surrounding surviving cells. Many of the remaining Purkinje cells show a hyaline necrosis. Magnification × 100.

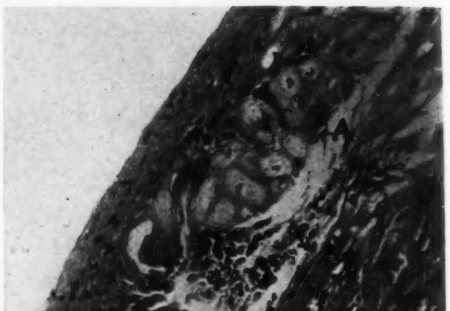

FIG. 16.—Pig No. 4. Subendocardial Purkinje strand anterior wall of left ventricle. (A) Normal cells. (B) Purkinje cells showing early hyaline necrosis associated with cellular infiltration. * This cellular infiltration is seen to produce complete interruption of the Purkinje strand. Magnification × 165.

alone would produce these effects: histological examination of the hearts of pigeons dead from chronic thiamin deficiency showed necrosis of the myocardial fibres associated with an inflammatory cell infiltration. De Soldati (1939) in thiamin deficient dogs noted high P waves and changes in the T waves and S–T segment.

In thiamin deficient pigs Wintrobe *et al.* (1943) recorded bradycardia, marked sinus arrhythmia, disturbances of A-V conduction and changes in the T waves. Bradycardia was considered to be a more pronounced degree than could be accounted for by inanition. Eight of the nine pigs showed prolongation of the P–R interval, two developed second degree block, and one complete heart block. The most constant T wave change was inversion of T IV. Atropine caused disappearance of a second degree heart block in one pig and in another shortening of the P–R interval and return to normal of the inverted T IV. In both pigs atropine caused an increase in the heart rate. The lesions found at autopsy in these animals included cardiac hypertrophy and dilatation, cellular infiltration, and scarring, as well as fresh necroses in the auricles and ventricles.

King and Sebrell (1946) in experimental thiamin deficiency in rats noted marked bradycardia, progressive widening of the P–R interval and QRS complex and an increase in amplitude of QRS complex and of the T waves in leads II and III. Auricular fibrillation and A-V nodal rhythm were noted in some animals during episodes of acute deficiency. Fourteen of twenty-four experimental rats showed cardiac lesions at autopsy; in ten of these the lesions were predominantly auricular.

Ashburn and Lowry (1944) in a detailed pathological study of thiamin deficient in rats found a moderate to marked dilatation of the right auricle; in some hearts the left auricle was dilated, but to a much less degree, and an occasional heart showed dilatation of the right ventricle. Histological lesions in the auricles were found in 46 of the 58, but ventricular lesions in only 7 of the deficient animals. The initial change consisted of a hyaline necrosis of the muscle cell associated with a cellular reaction consisting of neutrophils, lymphocytes, and mononuclear cells. There was ultimately disappearance of muscle fibres. Some of the acute lesions were followed by fibroblastic proliferation. The inactive lesions showed absence of muscle fibres and marked thinning of the auricular wall with some fibrosis. In a few instances there was a gross fibrosis of the

auricular wall. Van Etten *et al.* (1940) in thiamin deficient pigs described myocardial lesions consisting of scattered areas of atrophy and necrosis of muscle suggestive of infarction. The lesions were particularly marked in the wall of the left ventricle.

Porto and de Soldati (1940) found dilatation of the right auricle in four dogs maintained on a thiamin deficient diet. Two also showed dilatation of the right ventricle. Histological examination showed dissociation of the muscle fibres by interstitial oedema, lack of clarity of the transverse striations, hydropic degeneration of the muscle cells, most marked in the conducting system and areas of hyalinization of muscle fibres associated with cellular infiltration. The lesions were found disseminated throughout the heart, but were most marked in the right auricle and right ventricle. Follis *et al.* (1943) studied the lesions in thiamin deficient pigs. The hearts of six of nine animals were dilated and showed lesions consisting of focal or diffuse necrosis of the myocardial fibres associated with leucocytic infiltration. In an animal dying, early lesions were found in the auricles, but none in the ventricles. Animals that survived longer periods of deficiency showed lesions on both auricles and ventricles. Scars marking healed necrotic lesions were found in the hearts of two animals.

Swank *et al.* (1941) in five dogs dead from thiamin deficiency found dilatation of the right auricle and right ventricle in one; the others showed marked dilatation of the left side of the heart. Histological study showed some general shrinkage, and pale indistinct staining of the muscle fibres in all hearts. In three hearts small scattered areas of myocardial necrosis were observed, many of which were infiltrated with polymorphonuclear leucocytes. One heart showed older lesions consisting of small areas of loose connective tissue from which myocardial fibres were absent.

From these accounts of animal experiments it is apparent that morphological lesions in the myocardium may result from thiamin deficiency. There is, however, no complete agreement as to their character of distribution. In the experiment recorded the finding of dilatation of the right side of the heart agrees with the observations of Porta and de Soldati (1940) and of Ashburn and Lowry (1944). The increase in heart-body weight ratio above the normal 0·3 to 0·4 per cent found in this experiment has been noted by Follis *et al.* (1943). These authors point out however that it cannot be concluded from this, that the hearts were hypertrophied since in the presence of impaired growth from causes other than thiamin deficiency there is an alteration in the heart-body weight ratio.

The preponderant involvement of the auricular myocardium in this study agrees with the findings of Follis *et al.* (1942) in pigs, and those of Ashburn and Lowry (1944) and of King and Sebrell (1946) in rats. It is probable that the sinus block observed in three of the animals was related to morphological lesions in the auricular wall since it was unaffected by atropine. In the one animal in which it never appeared lesions in the right auricle were minimal.

This predilection of the myocardial lesions of thiamin deficiency for the auricles suggests that the metabolism of the auricular and ventricular myocardium differs in some respects. A difference in their metabolism has been suggested by Davies and Francis (1946) on the basis of a difference in their intrinsic rhythmic rates. These authors write "In the hearts of cold blooded vertebrates in which there is neither specialized muscular tissue (nodal and Purkinje) nor any histological difference in the ordinary cardiac muscle which later nevertheless exhibit different intrinsic rhythms when separated from each other, their different rhythmicities must be dependent on factors other than purely morphological characters." Davies and Francis (1946) have attempted to discover whether substances known to be concerned in the chemistry of contraction of voluntary muscle are unevenly distributed throughout the heart. They have shown that glycogen, phosphocreatine and adenosine compounds have such a differential distribution.

It is notable that a morphological lesion is not suggested as the causative factor in those instances in which disturbances of auriculo-ventricular conduction have been found. Thus Swank and Bessey (1942) minimize the significance of heart block in pigeons since they found that it might result from inanition alone; and Wintrobe *et al.* (1943) found that heart block in thiamin deficient pigs responded to atropine.

The experiment recorded shows conclusively that heart block and morphological changes in the cells of the conducting system may result from thiamin deficiency. It would appear that initially the block is due to a reversible biochemical disturbance, and that this disturbance is capable of progression to an irreversible morphological change if acute deficiency is of sufficient duration. The presence of histological lesions in a proportion of the cells of the bundle of His in animals No. 1 and 4 suggest that cardiograms taken immediately before the death of these animals would have shown heart block.

The selective involvement of Purkinje tissue found in this experiment suggests that these cells have also a metabolism which differs from that of the ventricular myocardium. Consistent with this view are the observations of Shaner (1930) and of Davies and Francis (1941*a*). Shaner's studies in the ontogenetic

development of this system in the calf suggests that Purkinje fibres and ordinary cardiac muscle are separately developed from their respective undifferentiated mesodermal primordia. Davies and Francis (1941*a* and 1946), have failed to demonstrate any nodal or Purkinje tissue in a number of fish, amphibia and reptiles, and express the opinion that the S–A, A-V nodes, the A-V bundle and the terminal network of Purkinje fibres are neomorphic developments in animals and birds associated with the more rapid rate of heart in these homiothermal vertebrates.

The infrequency of heart block in human beriberi in contrast to the readiness with which it may be produced in experimental thiamin deficiency in pigs may be explained by the difference between the structure of the conducting system in man and that in ungulates. This difference has been stressed by Glomset and Glomset (1940).

The biochemical or morphological changes are most likely to be found in those cells of the conducting system in man that most closely resemble in structure the Purkinje cells in ungulates. The cells most closely resembling the Purkinje cells in ungulates are found in man at the terminal ramification of the conducting system beneath the endocardium of the ventricles. As the conducting system is traced back toward the A-V node the similarity diminishes. It has been noted that Glomset and Glomset found no cells resembling the Purkinje cells of sheep or pigs in the bundle of His in man.

In thiamin deficiency of moderate degree abnormalities of the ventricular complex of the cardiogram probably result from changes in a proportion of the cells of the terminal ramifications of the conducting system.

It further follows that disturbances affecting conduction in the main branches of the bundle of His in beriberi would be of commoner occurrence than A-V block. It is, therefore, of interest that in one of the few reports of cardiac conduction defects in beriberi, that of Dock (1940), three of five cases showed bundle branch block and one latent A-V block.

Summary

The clinical impression of the not infrequent occurrence of auriculo-ventricular block in beriberi is recorded.

An experiment with the object of studying the effects of thiamin deficiency on cardiac conduction is described. Four pigs deprived of thiamin developed heart block; in two this was complete.

The histological lesions resulting from thiamin deficiency predominantly involve the auricular myocardium and the cells of the conducting system in the ventricles. In pigs dying from thiamin deficiency an acute hyaline necrosis is seen in the Purkinje cells.

I am indebted to Professor J. H. Biggart, C.B.E., and Dr. S. B. Boyd Campbell for the facilities that made this investigation possible. I wish to thank Professor Biggart for much advice and help in preparing this paper, and Dr. J. E. Morison for this valuable advice on the interpretation of the histological lesions. I am indebted to Mr. J. Harland for technical assistance and to Mr. D. McA. Mehaffey, A.R.P.S., for the photography.

References

Aalsmeer, W. C., and Wenchebach, K. F. (1929). *Wein. Arch. inn. Med.*, **45**, 1.
Ashburn, L. L., and Lowry, J. V. (1944). *Arch. Pathol.*, **37**, 27.
Blankenhorn, M. A. (1945). *Ann. intern. Med.*, **23**, 398.
Carter, C. W., and Drury, A. N. (1929). *J. Physiol.*, **68**, 1.
Casanova, J. (1946). *Algerie Med.*, **3**, 203.
Davies, F., and Francis, E. T. B. (1946). *Biol. Rev.*, **21**, 173.
——, ——, (1941*a*). *Philos. Trans. B.*, **231**, 99.
Dock, W. (1940). *Trans. Ass. Amer. Phys.*, **55**, 61.
Dustin, C. C., Weyler, H., and Roberts, C. P. (1939). *New Engl. J. Med.*, **220**, 15.
Van Etten, C., Ellis, N. R., and Madsen, L. L. (1940). *J. Nutrit.*, **20**, 607.
Follis, R. H., Jr., Miller, M. H., Wintrobe, M. M., and Stein, H. J. (1942). *Amer. J. Path.*, **19**, 341.
Glomset, D. J., and Glomset, A. T. A. (1940). *Amer. Heart J.*, **20**, 677.
Jolliffe, N., Goodhart, R., Gennis, J., and Cline, J. K. (1939). *Amer. J. med. Sci.*, **198**, 198.
Keefer, C. S. (1930). *Arch. intern. Med.*, **45**, 1.
King, W. D., and Sebrell, W. H. (1946). *Pub. Health Rep. Wash.*, **61**, 410.
Porto, J., and de Soldati, L. (1940). *Rev. Soc. Argent. Biol.*, **15**, 303.
Shaner, R. F. (1930). *Anat. Rec.*, **44**, 85.
de Soldati (1939). *Rev. Soc. Argent. Biol.*, **15**, 142.
Swank, R. L., Bessey, O. A. (1942). *Arch. intern. Med.*, **70**, 763.
——, Porter, R. R., and Yeomans, A. (1941). *Amer. Heart J.*, **22**, 154.
Weiss, S., Haynes, F. W., and Zoll, P. M. (1938). *Amer. Heart J.*, **15**, 206.
——, and Wilkins, R. W. (1937). *Ann. intern. Med.*, **11**, 104.
Wenckebach, K. F. (1934). *Das Beriberi-Herz: Morphologie, Klinik, Pathogenese. Pathologie und Klinik in Eingeldarst*, Band VI. (Springer).
Wintrobe, M. M., Stein, H. J., Miller, M. H., Follis, R. H., Jr., Najjar, V., and Humphreys, S. (1942). *Bull. Johns Hopkins Hosp.*, **71**, 141.
——, Alcayaga, R., Humphreys, S., and Follis, R. H., Jr. (1943). *Ibid.*, **73**, 169.

CARDIAC PAIN WITH RECOVERY OF THE T WAVE

BY

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Received September 6, 1948

Surprisingly little attention has been paid to the possibility of a return to normal of cardiograms that have been thought to show the presence of cardiac infarction when the clinical picture was consistent with that accident having occurred. It is, of course, well known that very transient cardiographic changes, to be measured in minutes or hours, may accompany an anginal seizure. But the authors are concerned not with these cases, but only with those in which the changes persisted for days, weeks, or even months, but ultimately disappeared. The return to normal was so complete that from the inspection of the cardiogram alone, including unipolar exploration, it was impossible to tell that anything abnormal had ever occurred. Careful search of papers published in the past twenty years and more has failed to reveal any such similar investigation, although recently, as a result of experiments on dogs, the possibility has been hinted at by Bayley and Monte (1943) that perhaps in man similar temporary changes might be found, which were not due to an actual infarct.

In every case the account of the pain left no doubt that it was of cardiac origin, and only such cases as showed inversion of the T wave, in leads I or IV or both, or pathological inversion of the T wave in lead III, were included in the series. In all cases standard leads and either IVR or CR 4, and IVF or CF 4, or both, were taken; and in all the patients encountered or followed up during the past two years unipolar leads were taken from the right arm (VR), left arm (VL), left foot (VF) and across the chest (V 1–6). The unipolar limb leads were augmented, using the Goldberger technique. Whenever the unipolar deflections obtained from the præcordium appeared inconveniently large in amplitude the fibre sensitivity was reduced by one-half, (N/2), or even to one-third (N/3). Although in our records the amplitude of the T wave is only one-half or one-third of the size of the usual deflection, the proportion of the height of this wave to that of the QRS complex remains, of course, unaltered. Some patients were further explored in a manner suggested by the results obtained from the above leads. For example, when the lateral wall of the left ventricle was involved, leads were taken from intercostal spaces higher than those usually employed, and in some of the cases suggesting posterior cardiac infarction a deep epigastric lead was taken. In all, the 28 cases were cardiographed on 140 occasions.

Selection of Cases and Frequency

We had constantly in mind such other causes as might lead to temporary inversion of T waves, and we took care to exclude these as far as possible. For example we excluded any patient in whom one might have suspected that digitalis was responsible for the T wave changes. All records were taken with the patient recumbent. There was no question of any patient having drunk cold water just before his test. None was receiving adrenalin; and, although one was diabetic, it was thought that insulin did not influence the T wave. One patient was myxœdematous, but the changes in the T wave were not characteristic of that disease: we are of opinion that it was not responsible for subsequent cardiographic changes. We excluded one patient with severe anæmia and two who were receiving desoxy-cortico-sterone acetate, for we did not consider that the changes in their T waves should find a place in this series. If there was any doubt concerning the presence of attacks of paroxysmal tachycardia, that case also was omitted. All the cases were adults, so the negative T waves of childhood were excluded. There was no question of trauma to the chest wall or elsewhere.

In the twelve-year period 1936–47 inclusive, cardiograms were taken from 5593 patients, and of those 367 yielded curves considered diagnostic of cardiac infarction. Of the 367 cases, 232 were of the anterior type, 131 were posterior, and 4 were considered to show evidence of both. Of the 232

anterior variety, 26 (11 per cent) were found that reverted to normal; but of the 131 posterior, only 2 (1·5 per cent) could be found whose curves had done so. None of the 4 cases showing combined anterior and posterior infarction resumed a normal appearance.

It is seen that of the total number of 367 cases of cardiac infarction, 28 (8 per cent) eventually became normal, and that of these there was a striking preponderance of the anterior type. Of these 28 cases, 22 have cardiograms that have remained free from any pathological change whatever during the time we have kept them under observation.

Clinical Details

Of our 28 cases, 22 were men and 6 were women. The ages when the patients were first seen ranged from 39 to 77, more than half of them being over the age of 50, and three were over 70. The average age was 54 years.

Although the period during which the patients were observed was in one case only two months, we were fortunate to have seven of our patients under observation for more than eight years. On the average they were followed up for just over four years. A study was made, from a consideration of the history and the type of the pain experienced, to see whether any clinical feature might emerge that would suggest the temporary nature of the abnormality in the cardiogram; but none did. The diagnosis of angina pectoris or cardiac infarction had been made in every case before the cardiogram was taken, and the curve was thought at that time to be confirmatory. In ten cases the pain was experienced only whilst the patient was at rest, while in seven pain was only present on exertion. In the remaining eleven patients pain was present both at rest and on effort. The pain recurred subsequent to the initial attack in 23 of the 28 patients. It was a peculiar feature that in five patients, in spite of recurrent and apparently typical cardiac pain, which in each of the five cases lasted more than an hour, the cardiogram remained normal. We could find no correlation between the severity or duration of the pain and the degree of inversion of the T wave. A persistently high blood pressure, the systolic being more than 160 or the diastolic greater than 95 or both, was present in 18 patients. In 9 it was normal and in one we have no record. Unfortunately we have insufficient information concerning serial white cell counts and sedimentation rates in this series to discuss their diagnostic value, but sometimes there was a slight degree of pyrexia, and the white cells and sedimentation rate were increased somewhat, although for the most part these were unchanged.

Cardiograms

Six facts emerged from inspection of the curves of this series. First, the anterior lesion was very much commoner than the posterior, much more so than with established infarction. Only 2 of the 28 cases were of the posterior type. Secondly, in every cardiogram showing an anterior lesion the Q wave was either absent altogether, or, if present, was very small. In no case was the Q wave in the lead or leads showing inversion of the T wave significantly prolonged (0·04 sec. or more); in lead I it did not exceed 1·0 mm. in depth, and in lead IV 1·5 mm. As a rule there was no Q wave (see Table). Thirdly, in the posterior type, unlike in the anterior, a Q wave may appear which is pathologically deep and wide in leads II, III, and VF, and yet ultimately this may revert to normal dimensions or disappear (Fig. 5). Fourthly, significant displacement of the RS–T segment by the current of injury was only present in two cases (Fig. 8) and was actually less than 2 mm. As the records were taken in several of our cases at all stages of the event, even during the paroxysm of pain, the absence of this current was surprising and will be commented on later. Fifthly, it was noted that once the cardiogram had reverted to normal the tendency was for it to remain so, and only in an unexpectedly small number did it deteriorate again. Thus, of the 28 cases that reverted to normal, 22 were still without pathological change when we last saw them. Of the other 6, one showed digitalis effect only, one showed the picture of left ventricular predominance, and in three of the remaining four that subsequently deteriorated, the abnormal feature was nothing more than an isoelectric T wave in lead I. The cardiogram of the fourth patient, after reverting to normal several times, finally persisted as a curve of cardiac infarction (Fig. 10). Finally, on several occasions when our records were taken, the patient was experiencing a severe attack of pain, but they did not necessarily show inversion of the T wave at that moment, although this abnormality subsequently appeared.

Prognosis

This is beyond doubt much better than that usually given for cardiac infarction. Of the 28 cases only 3 are dead and one of these (Case 1) died of septicæmia. Autopsy was performed and the findings are given below. No details are known of the mode of death of the other two, but one lived for 8 years following the appearance of his abnormal record and was 70 at the time of his death; the other died at the age of 79, his abnormal record having appeared when he was 75.

The exact length of time taken for the abnormal curve to revert to normal is difficult to assess

accurately unless records are taken at very frequent intervals. The times estimated for the curves to become normal are thus necessarily maximal. The shortest time was two days and the longest was three years and eight months, the average being 33 weeks. Of the 28 cases, 20 were active when last seen and, of these, 15 were quite free from pain. Three had experienced very slight pain which was inframammary in type and not of cardiac origin. Two patients had slight cardiac pain, with but little limitation of activity. Three others were free from pain but restricted by dyspnœa. Two were considerably hampered by pain: as already stated, three had died.

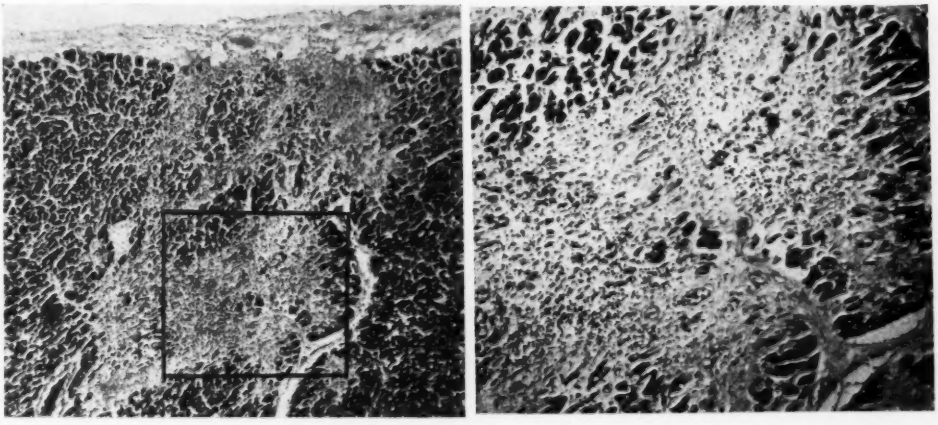
Morbid Anatomy

As mentioned above, the outlook in these patients would appear to be so good that opportunities for autopsy are few. The only one of this series (Case 1) that came to necropsy died as a result of obscure septicæmia. There was some excess of fat in the epicardium and the heart was enlarged and dilated, particularly on the right side. The myocardium was soft and flabby. Two doubtful pale areas of scarring, superficial and just visible to the naked eye, were present a little distance from the tip of the anterior wall of the left ventricle. In spite of careful search, section of the heart failed to reveal any other abnormal areas. The main coronary vessels were quite patent and quite free from atheroma. The valves and pericardium were normal. Early changes of right-sided heart failure were present in the lungs, liver, spleen, and kidneys.

Microscopically, the small sub-epicardial area sectioned consisted of a fairly well localized patch of fibrosis. Microscopic branches of the coronary vessels seen were healthy (Fig. 1).

Illustrative Cases

Unfortunately, space does not permit us to include full clinical and cardiographic details of all 28 cases, so we have listed the more important features (see Table) and chosen to illustrate from our series only such examples as show features worthy of particular comment. This has necessitated the omission of a number of illustrations showing widespread and well-marked abnormalities, but of similar form. Ten cases are presented: the first is one in which autopsy was performed; the second is a typical anterior lesion; the third is also anterior and shows how widespread abnormalities in the unipolar præcordial leads can revert to normal; the fourth is one of our two posterior lesions in which the cardiogram recovered; the fifth is an example of anterior type where only T 4 was inverted and not T 1; the sixth showed inversion of T 1 but not of T 4; the seventh is presented because it is one of the two in which any RS–T segment displacement occurred; the eighth case, although presenting a seemingly established picture, showed that by inhalation of amyl nitrite it was possible to elevate an inverted T 1 and T 2, although these two waves became again inverted after five hours; the ninth case shows spontaneous inversion of T 1 and T 4, often but not always associated with attacks of cardiac pain; the tenth case is



A B

Fig. 1.—Case 1. The larger of two very small pale areas just visible beneath the epicardium. It consists of a well localized patch of replacement fibrosis. The branches of the coronary vessels are quite healthy. (A) Magnification × 35. (B) Magnification × 105.

Case No. Sex Age*	Period under observation	PAIN		B.P. (Maximum)	CARDIOGRAM				Final state of the patient
		No. of attacks E = on effort R = at rest	Severity. Duration of longest attack		Max. depth of Q wave in mm.	Max. inversion of T wave in mm.	Final appearance	Time taken for changes to disappear	
1. M, 43	4 months	E. R. **	Moderate. " Strangling." 20 min.	160/100	Q 1 absent Q 4 absent	T 1 = −1·5 T 4 = −3·0	T 1 and T 4 isoelectric	3 months	Died of septicæmia. Autopsy
2. M, 53	10 years	E. R. **	Severe. " Violent." 10 min.	150/130	Q 1 absent Q 4 absent	T 1 = −1·5 T 4 = −2·5	Normal	44 months	Active. No pain
3. M, 47	1 year	E. R. 4	Extremely severe. " Pulling together." 20 min.	130/90	Q 1 absent Q 4 absent	T 1 = −1·5 T 4 —	Normal	1 month	Active. No pain
4. M, 48	4 years	R. 1	Very severe. " Indigestion." 4 hours	128/80	Q 2 = 2·0 Q 3 = 3·0	T 2 = −3·0 T 3 = −5·0	Normal	2 months	Active. No pain
5. M, 66	9½ years	E. 3	Severe. " Oppression." All day	195/100	Q 1 = 1·0 Q 4 absent	T 1 flat T 4 = −6·5	Normal	5 months	Active. No pain
6. M, 43	3 years	R. 2	Moderate. " Sore trachea." Several hours	130/80	Q 1 = 0·5 Q 4 absent	T 1 = −2·0 T 4 = +1·0	Normal	3 months	Active. No pain
7. F, 53	8 months	E. R. 4	Very severe. " Suffocating constriction." Several hours	180/110	Q 1 = 0·5 Q 4 absent	T 1 = −1·0 T 4 = −2·5	Normal	2 months	Active. Considerable pain
8. F, 54	6 months	E. R. **	Severe. " Indigestion." 20 min.	200/140	Q 1 absent Q 4 absent	T 1 = −2·0 T 4 = −4·5	L.A.D. only	4 months	Restricted. Considerable pain
9. M, 50	9½ years	E. R. **	Severe. 2 hours 40 min.	186/120	Q 1 = 0·5 Q 4 absent	T 1 = −1·5 T 4 = −5·0	Infarction	Few days	Restricted by dyspnœa. No pain
10. M, 63	4 years	R. 1	Very severe. " Burning." Several hours	200/105	Q 1 absent Q 4 absent	T 1 = −1·0 T 4 = −2·5	Digitalis effect only	2 days	Restricted by dyspnœa. No pain
11. M, 45	10 years	R. 2	Extreme. " Pressure or tightness." 1 hour	160/100	Q 1 absent Q 4 absent	T 1 = −3·0 T 4 = −6·0	Normal	5 months	Active. No pain
12. F, 77	3 years	R. **	Severe. " Angina." 20 min.	180/100	Q 1 absent Q 4 absent	T 1 = −1·5 T 4 = −2·0	Normal	38 months	Active. No pain
13. F, 52	8 years	E. R. **	Severe. " Painful tightness." Indefinite	150/100	Q 1 = 0·5 Q 4 absent	T 1 = −2·0 T 4 = −5·0	Normal	17 months	Active. Occasional submammary stabbing pain only
14. M, 52	1 year	R. 1	Severe. — Unknown	Unknown	Q 1 absent Q 4 absent	T 1 = −1·0 T 4 = −2·0	Normal	3 months	Active. No pain

Case No. Sex Age*	Period under observation	PAIN: No. of attacks E = on effort R = at rest	PAIN: Severity. Duration of longest attack	B.P. (Maximum)	CARDIOGRAM: Max. depth of Q wave in mm.	CARDIOGRAM: Max. inversion of T wave in mm.	CARDIOGRAM: Final appearance	CARDIOGRAM: Time taken for changes to disappear	Final state of the patient
15. M, 62	8 years	E. **	Severe. "Tightness." 5 min.	160/120	Q 1 absent Q 4=1·5	T 1= 0 T 4= −8·5	Normal	2 months	Died; aet. 79
16. M, 59	3 months	E. 1	Severe. "Severe." Half hour	165/90	Q 1 absent Q 4 —	T 1 upright but TV 1–6 all inverted	Normal	29 days	Active. No pain
17. F, 64	2 years	R. 3	Severe. "Gnawing." 4 hours	205/120	Q 1 absent Q 4 absent	T 1= −0·5 T 4= −2·0	Normal	17 days	Active. Only inframammary pain
18. M, 55	6 months	R. 2	Excruciating. "Gripping compression." Half hour	135/90	Q 1 absent Q 4 —	T 1= −0·5 T 4 —	Normal	5 weeks	Active. No pain
19. M, 39	1 year	E. **	Moderate. "Tightness." Few min.	140/92	Q 1 absent Q 4=1·0	T 1= −1·0 T 4= −2·0	Normal	2 months	Active. No pain
20. M, 42	4 years	E. **	Moderate. "Heaviness." ¼ hour	118/82	Q 1 absent Q 4 absent	T 1= −1·5 T 4= −12·0	T 1 isoelectric	5 months	Active. Pain on exertion
21. M, 75	2 years	E. R. **	Moderate. "Tight gripping." 1½ hours	155/80	Q 1 absent Q 4 absent	S–T 1 seg.= −1 S–T 4 seg.= −2	Normal	1 month	Died; aet. 79
22. M, 48	3½ years	E. R. **	Moderate. "Aching." 20 min.	160/110	Q 1 absent Q 4 —	T 1= −1·5 T 4 —	Normal	1 month	Active. No pain
23. M, 42	4 years	E. R. **	Very severe. "Crushing." 4 min.	130/90	Q 1 absent Q 4 absent	T 1= +0·5 T 4= −2·5	Normal	5 months	Restricted. Considerable pain
24. F, 55	6 years	E. R. **	Severe. — 2 hours	210/120	Q 2 absent Q 3=2·0	T 2= −1·5 T 3= −4·5	Normal	17 months	Active. Slight pain
25. M, 55	10½ years	E. **	Moderate. "Indigestion." Few min.	140/100	Q 1 absent Q 4 absent	T 1= +0·5 T 4= −1·5	Normal	15 months	Active. No pain
26. M, 39	2 months	E. **	Severe. "Tightness." 5 min.	160/120	Q 1 absent Q 4=1·5	T 1=0 T 4= −8·5	Normal	2 months	Active. No pain
27. M, 67	10 years	R. 2	Severe. — 20 min.	180/95	Q 1 absent Q 4 absent	T 1= −2·5 T 4= −9·5	Normal	10 months	Active. No pain
28. M, 75	1 year	R. 1	Severe. — 18 hours	164/95	Q 1 absent Q V4 absent	T 1= −2·0 T V4= −10·0	T 1= −0·5 T V4= +3·5	1 year	Restricted by dyspnœa. No pain

* Age when patient first came under observation.
** More than six distinct attacks of pain at rest, or persistence of exertional pain.
All lesions were anterior excepting cases 4 and 24 which were posterior.

included because it is the only example we have met where the T wave in lead CF 4 varied, alternately being upright and inverted from beat to beat.

Case 1 (*Fig.* 1 *and* 2). *Man, aged* 43. In July 1946 typical symptoms of failure of the left ventricle first appeared. Dyspnœa on effort was soon followed by attacks of severe nocturnal dyspnœa. A few weeks later he complained of a choking sensation, as though he were being strangled, accompanied by a dull pain in the left chest. This pain was at first only brought on by exertion such as walking and always brought him to a standstill. It lasted for fifteen to twenty minutes, never longer, and did not radiate.

During his stay in hospital several of these attacks of pain occurred whilst he was asleep, and awakened him. Auricular fibrillation was present intermittently, and, although on admission his blood pressure was 160/100, it fell during his stay to 130/90 and, just before death, to 80/48.

Whilst in hospital he developed a fatal septicæmia, the origin of which was never discovered.

On 29/10/46 his cardiogram confirmed that auricular fibrillation was present. T 1 and T 2 were flat and T 4F was sharply inverted. By 6/11/46, T 1 was inverted, and inversion of T 4 persisted. No Q waves were present. Further cardiograms taken on 15/11/46 and 2/12/46 showed some improvement, and by 28/1/47 normal rhythm was present, T 1 was flat and T 4 was almost so.

Comment. This case is presented in some detail as we were fortunate enough to obtain a necropsy owing to the patient's death from septicæmia. Although the last cardiogram (28/1/47) before his death on 26/2/47 was not normal, it showed considerable improvement over the one taken on 6/11/46, which was suggestive of anterior cardiac infarction. The inversion of T 1 and T 4 had practically disappeared within the last three months. Naked eye and microscopical appearances have been described and illustrated above (Fig. 1). Notwithstanding the cardiographic appearances, the degree of localized cardiac muscle destruction was minimal. We feel here that, had the patient not succumbed to incidental disease, such a trivial lesion would hardly have impaired a very good chance of complete recovery, particularly in the absence of coronary atheroma.

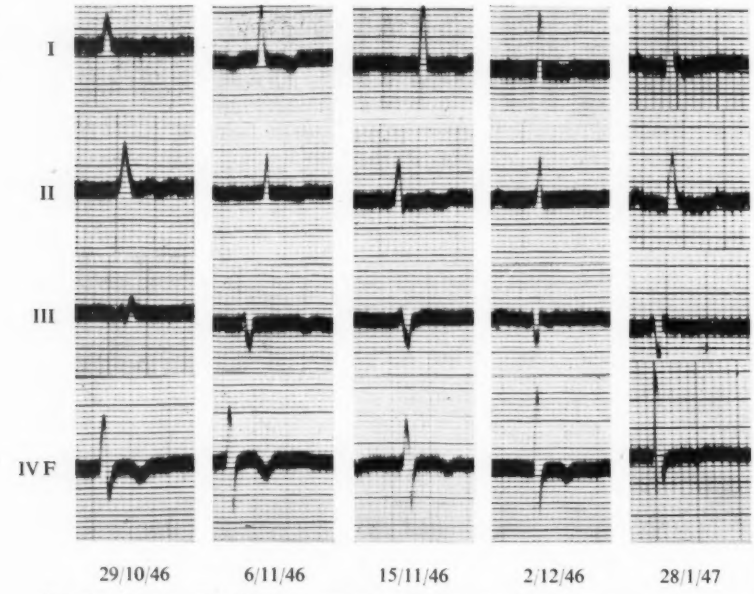


FIG. 2.—Case 1. Cardiogram suggesting anterior cardiac infarction, and showing improvement prior to death from septicæmia. Auricular fibrillation.

Case 2 (*Fig.* 3). *Man, aged* 53. The patient was quite well until 23/3/38 when he suddenly experi-

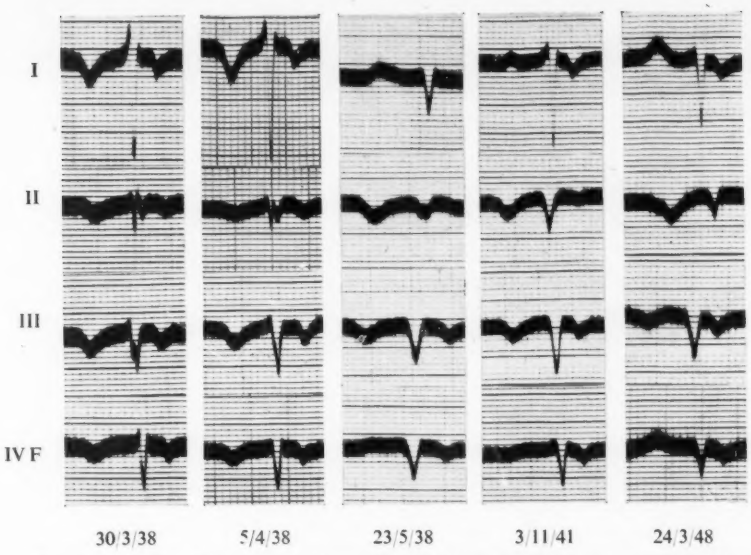


FIG. 3.—Case 2. Recovery of abnormal cardiogram suggesting anterior infarction but with no Q waves.

enced a violent substernal pain whilst he was at rest. Subsequently this type of pain occurred on exertion, the attacks lasting five to ten minutes and being relieved by rest. Whenever the pain was particularly severe it radiated down his left arm. When seen six days after his first attack of pain, his blood pressure was 118/95, gallop rhythm was present, and he was found to have diabetes.

His cardiogram on 30/3/38 was taken during an attack of pain and showed inversion of T 1 and T 4F and elevation of T 3. The R waves were of low voltage but no Q waves were present. When repeated on 5/4/38 T 1 had become flat and T 4 had become 2 mm. less inverted. By 23/5/38 the gallop rhythm was still faintly audible. His cardiogram was virtually the same.

When seen on 13/6/38 he was still experiencing occasional pain, the gallop rhythm was gone, and he could walk a mile or so before becoming breathless. His B.P. was 105/65. He was seen again on 3/11/41 and the cardiogram was taken just as an attack of pain had ceased. To our surprise the record was quite normal and even the R wave amplitude had increased. When seen on 29/5/46 he was experiencing rather vague " neuritic " præcordial pains on walking, there were no abnormal physical signs, his diabetes was controlled by diet alone and his cardiogram was still normal.

He was seen again on 24/3/48 and he had been keeping symptom-free, showed no abnormal physical signs, B.P. 140/90, and the cardiogram in the standard leads, unipolar limb and præcordial leads, was quite normal.

Comment. Typical cardiac pain was present, coming on at first at rest and later on exertion also, in a moderate diabetic. His cardiogram was originally considered typical of anterior cardiac infarction, becoming normal before the attacks of pain had ceased. In fact, one taken immediately after a severe attack of pain was quite normal. Ten years from the first attack, his cardiogram is still normal. This case illustrates well how a normal cardiogram may be consistent with the presence of severe cardiac pain.

Case 3 (*Fig.* 4). *Man, aged* 48. At the beginning of April 1947 whilst the patient was at rest in a chair, he had a sudden attack of pain and numbness which began in the left arm and radiated up to his left shoulder and across the upper part of the sternum, where it became of frightening intensity and was associated with considerable dyspnœa. The chest pain was described as " pulling together, as though the left arm were being torn out." Much *angor animi* was present. He sweated profusely during the attack and his wife commented on his extreme pallor. The attack lasted twenty minutes and went off quite suddenly. During the next two hours he had two

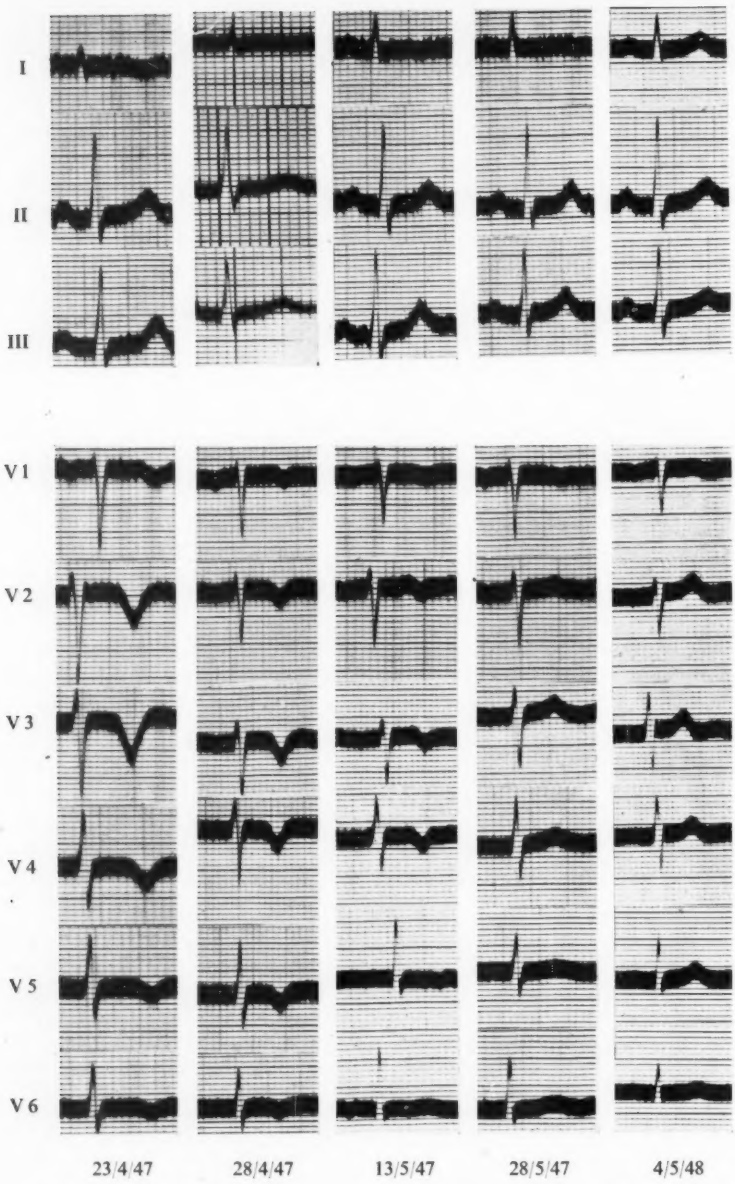


FIG. 4.—Case 3. Cardiogram suggests widespread anterior lesion, but without the presence of Q waves. The record became normal within five weeks, and a year later was still so. V lead fibre-sensitivity = N/2.

similar, but less severe, episodes whilst in bed. A further attack came on whilst he was walking from work and the pain again started in the left arm, which felt heavy.

Clinical examination on 23/4/47 revealed no abnormal physical signs, B.P. 130/90, but the cardiogram taken the same day showed, in addition to slight inversion of T 1 with a low voltage QRS, pathological T inversion of all the unipolar præcordial leads, especially V2, V3, and V4, although no Q waves were present. The B.S.R. taken the next day was 4 mm. in the first hour (Westergren) and a white cell count taken on 25/4/47 was 12,000 per c.mm. It was considered that the patient had had a coronary artery occlusion, but that although the ischæmic area was of great extent, it was not transmural.

He was treated with anti-coagulant therapy.

No further pain occurred whilst he was in hospital and on the day of his discharge, 13/5/47, the cardiogram showed that his præcordial T waves had become less inverted, those of V5 and V6 being flat. A fortnight later, on 28/5/47, T 1 was just upright and V2–6 inclusive had become upright. He was last seen on 4/5/48 and he had been quite free from pain or disability of any sort for one year and his cardiogram, standard, unipolar limb and præcordial leads, was quite normal.

Comment. Cardiac pain of great severity was associated with cardiographic changes of extensive antero-lateral distribution without Q waves. These changes, together with the pain, disappeared within five weeks, and a year later, both clinically and cardiographically, the patient was quite normal. It is noteworthy that fever, leucocytosis, and increase in the sedimentation rate were inconspicuous.

Case 4 (*Fig.* 5). *Man, aged* 48. Whilst sitting at a desk at 10.30 p.m. on 14/7/44 the patient experienced sudden intense, continuous, retrosternal pain, which lasted four hours (" like very severe indigestion, although much worse "). He also felt it in both arms, from elbow to thumb, and it caused him to sweat. His doctor found no abnormal physical signs, although he saw the patient a few hours later. The patient felt absolutely fit within two days and he was sent to hospital where a cardiogram, taken on 20/7/44, only revealed a rather tall P wave in lead II, although there was slight inversion of T III. However, in view of his story he was admitted to hospital, and further cardiograms were taken. Although no further pain occurred, the second record taken on 31/7/44 showed the usual appearances of posterior cardiac infarction with deep inversion of T II and T III and prominent Q II and Q III. The cardiogram improved and by 18/9/44 it was quite normal.

When seen on 4/5/48, nearly four years after his attack, he had been keeping very well indeed and was leading an active life, without any pain or other disability whatsoever. There were no abnormal physical signs, B.P. 120/75, and his cardiogram was still quite normal.

Comment. A single isolated attack of severe cardiac pain associated with a cardiographic appearance that, although at first was normal, showed a few days later the typical appearance of posterior infarction, including the presence of Q waves. Even so, the cardiogram had recovered within two months.

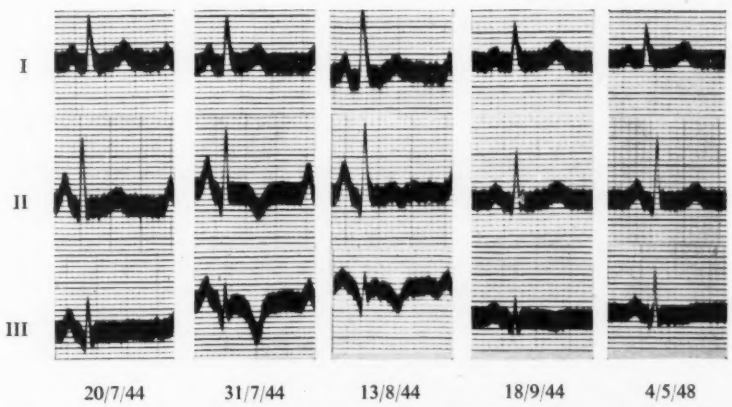


FIG. 5.—Case 4. A type of posterior lesion. The cardiogram recovered within two months, and both the patient and his record have remained normal for four years.

The patient has remained quite fit and has had a normal record for four years. This case also is an example of cardiac pain being accompanied for a few days by a normal cardiogram which only subsequently assumed the appearance typical of infarction.

Case 5 (*Fig.* 6). *Man, aged* 66. This active fisherman was first seen by us nearly ten years ago and complained of three distinct attacks of severe præcordial oppression which radiated down the left arm. The first occurred on 8/8/38, while he was pushing his boat down to the beach. The pain during the third attack lasted all day.

He was first seen on 5/9/38. There were no abnormal physical signs apart from a very slight scoliosis and a trace of albumen in the urine, B.P. 130/78. The cardiogram on that date showed T 1 to be rather low but upright, but deep inversion of T 4F was present, although there were no Q waves. A record taken on 24/10/38 showed normal standard leads, but T 4F was only just upright. On 27/2/39 his cardiogram was quite normal with an upright T in 4F of good amplitude, and a Q wave had appeared. The pressure had risen to 145/105.

A cardiogram on 23/5/39 was identical in appearance with that of 27/2/39. The pressure had risen to 160/110.

When last seen on 14/1/48 the blood pressure was 195/100. The cardiogram, standard leads, CR 4, CF 4, unipolar limb and præcordial leads, was quite normal and he was leading a normal symptom-free life ; during the whole of the war his work had been particularly arduous.

Comment. This is an example of cardiac pain precipitated by severe exertion, accompanied by changes in lead 4F but without changes in lead 1. Within six months the curves had become normal and they have remained normal for nearly ten years and the patient has remained well. Lenègre and Chevalier (1946) have recently recorded 32 patients with such T wave changes and concluded that provided the patient is more than twenty years of age, isolated inversion of T 4 is pathological and most often of coronary origin.

Case 6 (*Fig.* 7*A and B*). *Man, aged* 43. When first seen on 3/5/45 his history was that ten days previously he had felt ill and feverish for a day but did not go to bed. The next night he still felt unwell and woke at 11 p.m. with a nasty dull pain in the upper chest, which he described as being like a " sore trachea." He was restless during the pain, which lasted for several hours. The pain recurred, though less severely, at odd times during the next few days

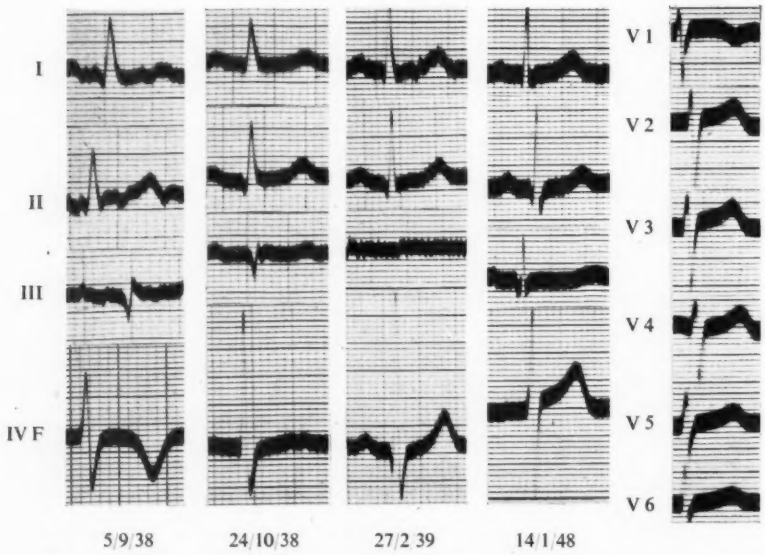


Fig. 6.—Case 5. An example of inversion of T 4F but with T 1 remaining upright. Cardiographic recovery occurred within six months and has persisted for nearly ten years. V lead fibre-sensitivity = N/2.

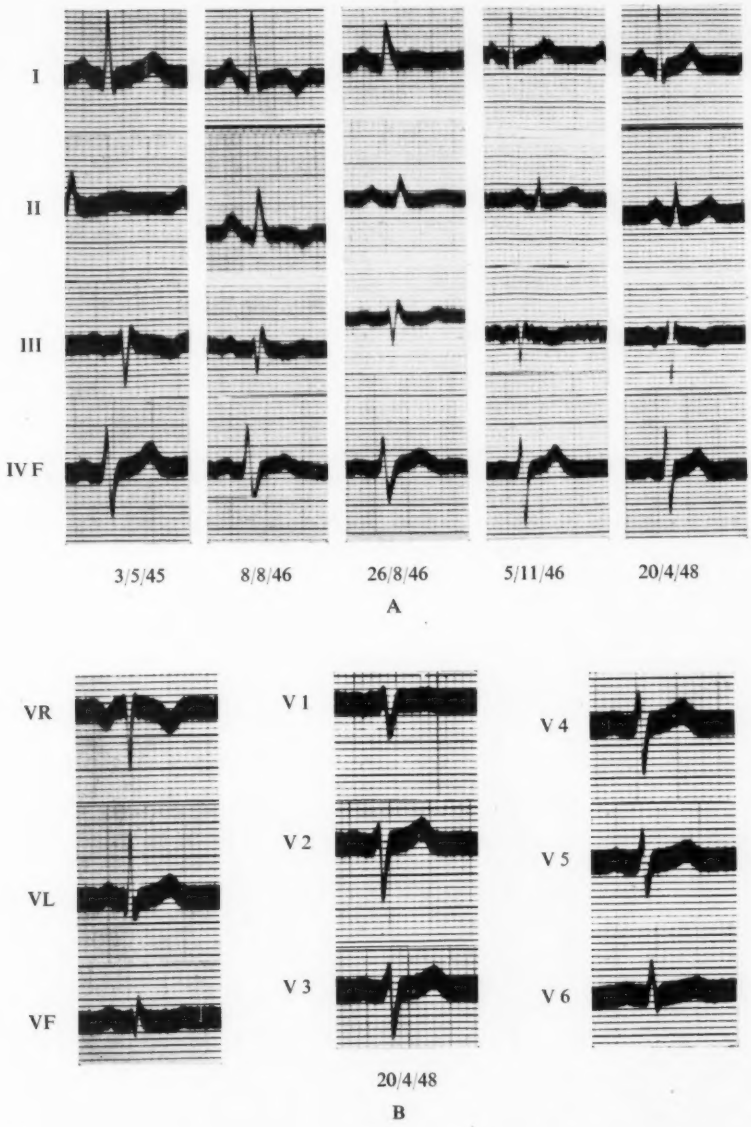


FIG. 7.—Case 6. An example of inversion of T 1, and T 2, but with T 4F remaining upright. The record reverted to normal within three months. (A) Standard leads and lead 4F showing deterioration and recovery. (B) Final unipolar leads (N/2).

and was then described as "constricted breathing," more a discomfort than pain.

There were no abnormal physical signs, B.P. 130/80, and cardioscopy was normal. His cardiogram taken the same day showed only a little left axis deviation. He remained well until 4/8/46 when a second similar attack occurred suddenly at 2 a.m. and lasted for several hours.

A record taken on 8/8/46 revealed that now T 1 and T 2 were both inverted, and although T 4F was not of good amplitude it was still upright. Within eighteen days T 1 had improved and become flat and T 2 was upright. By 5/11/46 the curves were normal, and when the patient was last seen on 20/4/48 he was keeping quite free from pain and leading an active life. His cardiogram was still quite normal in standard leads, 4F, unipolar limb, and præcordial leads.

Comment. An example of cardiac pain at rest, at first accompanied by a normal cardiogram and later by one in which T 1 and T 2 became inverted and yet T 4F remained upright. Within three months of the first abnormal cardiogram, his record had become normal. Here again, it would have been erroneous to assume that his normal cardiogram ruled out the possibility of his pain being cardiac in origin.

Case 7 (*Fig.* 8). *Woman, aged* 53. This woman had four quite severe attacks of cardiac pain, all coming on whilst she was at rest. The first attack woke her during the night of 8/9/47 and there was such severe substernal pain and dyspnœa that she thought she was going to die. The pain radiated down the left arm into the left middle finger. When seen next day the only abnormal sign was her blood pressure of 180/110. The second attack occurred at 6 a.m. on 11/9/47, was similar to the first and accompanied by much sweating, a pulse rate of 156, and a temperature of 99. The next day, 12/9/47, her cardiogram was taken (standard leads, V1, V4, VF, and VL) and was normal. White cell count on 13/9/47 was 5000 per c.mm. with a normal differential. B.S.R. on 16/9/47 was 12 mm. in the first hour (Westergren).

On 25/9/47 a third attack occurred. She had a little pain in her chest during the day and this suddenly increased and became intense and radiated down the left arm to the wrist. She felt hot and frightened. During the attack her blood pressure was 160/100 but next day had fallen to 120/98. A record taken on 26/9/47, the day following the third attack of pain, showed that the RS–T junction and segment in leads 1, 2, and V4 was raised and in leads 1 and 2 it incorporated the T waves. In V4 the T wave was inverted. The segment elevation was about 1 mm. in leads 1 and 2 and was considered consistent with anterior infarction, notwithstanding the facts that her white cell count taken the day after the attack was normal, 8·4 thousand, and her B.S.R. taken on 29/9/47 was 7 mm. in the first hour (Westergren).

By 15/10/47 no further attacks had occurred and the RS–T segment had gone back to the isoelectric level and, as we expected, T 1 and T 2 had become inverted in addition to T in V4. T 3 had also

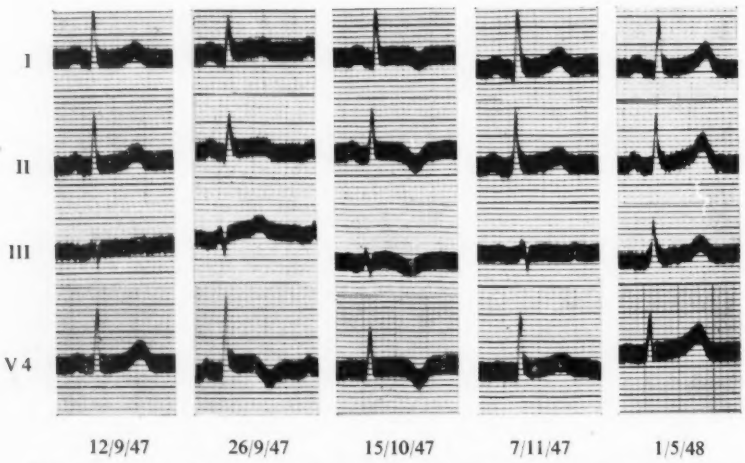


FIG. 8.—Case 7. One of the two records in the series which showed appreciable RS–T segment displacement (26/9/47). It disappeared in less than three weeks.

become inverted and this was thought to indicate an extension of the lesion. By 21/10/47 the curves had become quite normal and were repeated on 7/11/47 when they were still normal. A fourth attack of pain, felt as a constriction beneath the upper part of the sternum and radiating down the left arm to the elbow, occurred on 21/4/48, but a cardiogram taken on 1/5/48 which included standard leads, unipolar limb and præcordial leads, was still quite normal.

Comment. There are several reasons for presenting this case. It is one of only two cases in which we have been able to detect segment displacement, although this was only slight. Secondly, it again illustrates how it was not until three attacks of cardiac pain had occurred that any alteration in the cardiogram was detected. In spite of apparent extension of the lesion the cardiogram became normal within three weeks, and in spite of the fourth attack of pain, it remained normal.

Case 8 (*Fig.* 9). *Woman, aged* 54. This patient had a six weeks history of " indigestion " when first seen, which during the latter five weeks had been felt as a " terrible dull boring pain " beneath the lower sternum, going up into both jaws and down to both elbows. Without trinitrin the pain lasted twenty minutes. It came on with exertion and caused her to stop walking, an exercise of which she was previously very fond, and it also awakened her.

On examination on 5/12/47 her B.P. was 200/140 and a very clear presystolic gallop rhythm was visible, palpable and audible. A cardiogram taken the same day showed negative T waves in leads I, V1, V3, V4, V5, V6, and CF 4. T 2 was flat. No Q waves were present. These changes were consistent with antero-lateral infarction and hypertensive heart disease. Whilst she was in bed several more attacks of pain occurred and repeated B.S.R. estimations and white cell counts were made during and between these attacks. The highest B.S.R. was 10 mm. in one hour and the highest white cell count was 11·7 thousand per c.mm. with a normal differential count.

Cautiously, the effect on the cardiogram of exercise, of chewing trinitrin tablets, injections of tetraethyl ammonium bromide, and finally of inhaling 5 minims of amyl nitrite was observed. All except the last were without appreciable effect on her cardiogram. The result of amyl nitrite on 30/12/47 was striking. The previously inverted T 1 and T 2 became upright and T CF 4 was appreciably less inverted. Five hours later the cardiogram had regained its former pathological appearance.

By 2/4/48 all signs of infarction change had disappeared and only those due to left ventricular preponderance remained, the inverted T waves in

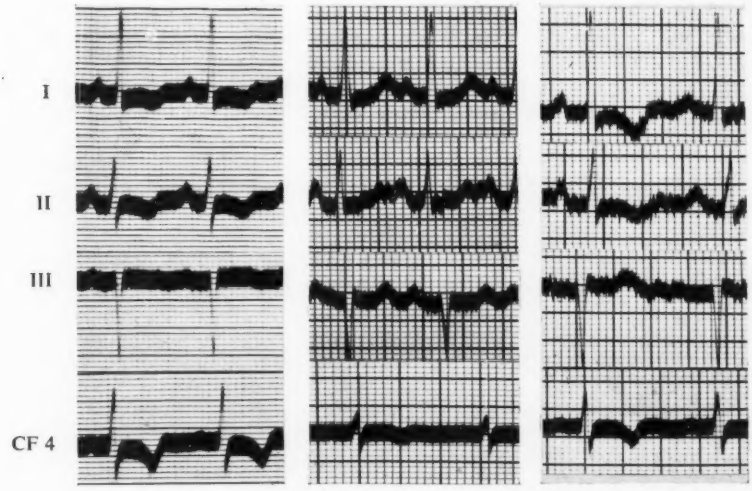


FIG. 9.—Case 8. Showing how by inhalation of amyl nitrite it was possible temporarily to elevate inverted T waves in leads I and II.

lead II having become upright and the deeply inverted T waves in V2–6 inclusive having all disappeared.

Comment. Severely incapacitating cardiac pain, both on effort and at rest, at first was accompanied by cardiographic changes of hypertensive heart disease and later by changes suggesting an extensive area of infarction, but without Q waves. By inhalation of amyl nitrite it was possible temporarily to cause elevation of the T waves in the standard leads. During the inhalation of amyl nitrite there was an increase in the heart rate, but this was no greater than had previously been produced by exercise without causing any alteration in the T waves.

Case 9 (*Fig.* 10). *Man, aged* 50. About the middle of February 1939 the patient began to experience attacks of severe pain just to the left of the sternum, passing down both arms. It came both on exercise and whilst he was asleep in bed. During each attack he became pale and a little cyanosed and was obviously in pain. The duration of each attack varied from half an hour to two hours forty minutes and the pain was best relieved by amyl nitrite. Between attacks he felt perfectly well, there was no dyspnœa, and his B.P. ranged from 130/86 to 140/96; on several occasions it was found to be raised immediately preceding an attack of pain, 160/110 to 186/120.

A teleradiogram revealed slight enlargement of the left ventricle with some elongation of the aorta. The first cardiogram taken on 10/3/39, two days after a very severe and prolonged attack of pain, showed T 4R to be sharply inverted, although no Q waves were present. T 1 was possibly a little low though T 2 was of good amplitude. A second record taken on 13/3/39, the patient having remained free from pain since his attack on 8/3/39, showed that T 4R had become quite upright. Records repeated on 14/3/39 and 27/3/39 showed T 1 and T 4 to be still upright.

Further attacks of pain occurred on 8/4/39, 11/4/39, and 13/4/39, and a record taken on 14/4/39 revealed that T 4 had again become deeply inverted and that T 1 had now become inverted for the first time.

He remained free from pain until 20/4/39, on which day a very severe attack occurred lasting two hours forty minutes and accompanied by pallor, sweating, and tic-tac rhythm. We obtained a record within a quarter of an hour of the start of this attack and it was normal, T 1 and T 4 having become upright since the previous record. We repeated his cardiogram on the day following the attack and T 1

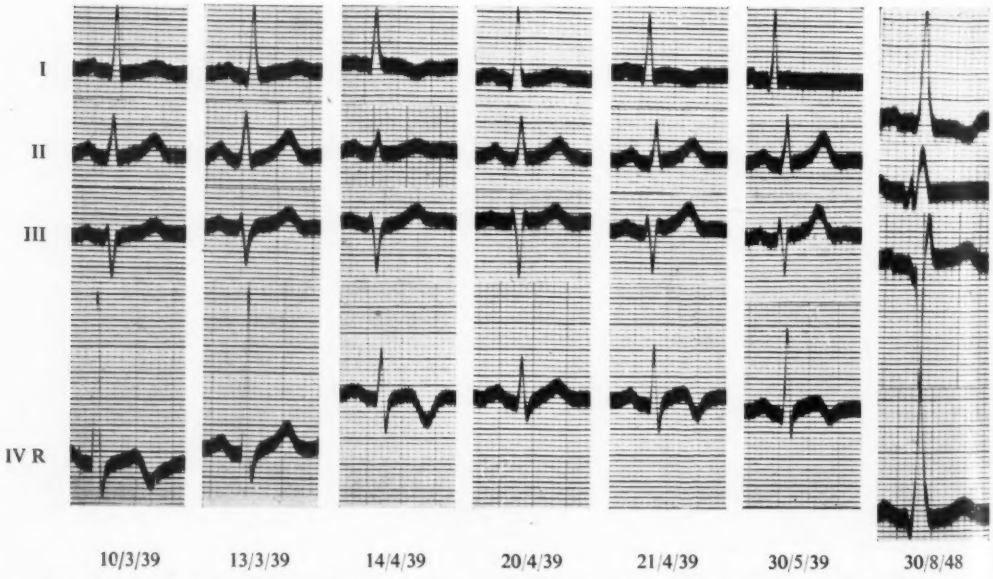


FIG. 10.—Case 9. Showing deterioration and improvement of cardiogram, of rapid sequence, until finally deterioration persists. This suggests that a transient myocardial defect, at first reversible, finally became established.

and T 4 were then found to have become inverted once more, and the appearance of the RS–T segment in 4R was strongly reminiscent of a recent current of injury curve. From that time on the curves remained abnormal. For example, a record was taken each day from 22/4/39 to 28/4/39, and on various dates until 30/5/39. On 23/4/39 half-hourly records were taken from 7 a.m. to 12.30 p.m., covering a period in which the patient was in pain and during which the pain wore off, but all records show the same inversion of the T waves.

It was at that time considered that the change had probably become permanent although the Q waves were insignificant. When seen again on 30/8/48 he stated he had remained free from pain since the middle of 1939, but that his breathlessness on exertion had lately become worse, and attacks of nocturnal dyspnœa were now occurring. His B.P. had fallen to 130/80, and although T in IVR had become upright a wide Q of 2 mm. depth had appeared in this lead, and T 2 had become flat (Fig. 10).

Comment. The records strongly suggest that a transient myocardial lesion, at first reversible, finally became established. Again the cardiographic deterioration lagged considerably behind the patient's pain.

Case 10 (*Fig.* 11). *Man, aged* 63. This was a case of hypertensive heart disease. On admission to hospital his B.P. was 195/115 and his rhythm, confirmed by cardiogram on 30/12/47, was normal, the curve showing only signs of left ventricular hypertrophy. Transurethral resection of the prostate was performed on 2/1/48, and the following day the patient complained of extremely severe burning pain in the chest and back. He was pale and sweating, and auricular fibrillation was present. The blood pressure had fallen to 110/80 and many pulmonary crepitations were audible. The pain lasted several hours and we obtained a cardiogram during the attack. This confirmed the presence of auricular fibrillation and lead CF 4 showed the T wave to be upright in some beats and inverted, occasionally sharply so, in others. At times an upright T wave alternated with an inverted one. The QRS deflection did not show this alternation.

He was immediately given digitalis and also oxygen and papaverine. By the next day he had regained normal rhythm and this persisted. A record taken on 5/1/48 confirmed the presence of normal rhythm, and by that time the T waves in CF 4 showed the effect of digitalis only and were all of similar form and amplitude. At the time of his discharge from hospital his B.P. was 200/105.

Comment. Sudden severe pain associated with much shock and transient fibrillation occurred on the day following prostatectomy. During this severe pain the T waves in CF 4 showed alternation which had disappeared two days later when normal rhythm had reappeared. No Q waves were present. The only other case we could find illustrated with alternation of the T wave from upright to inverted, in that case in lead III only, is the one recorded by Hamburger, Katz, and Saphir (1936). Their patient, a woman aged 48, suffered from anginal attacks and at autopsy small multiple recent and organizing infarcts were present.

Discussion

The problem is to determine the nature of the underlying pathological state that gives rise to the cardiographic changes described. Any attempt to solve it must be largely conjectural, as the condition is of such good prognosis that it rarely presents an opportunity for autopsy.

Inversion of the T wave can occur in the apparently normal subject, as White (1945) has pointed out. We therefore first considered whether perhaps some of our cases might be accounted for in this way, although the presence of cardiac pain, and the frequently associated hypertension, can scarcely be considered as incidental findings. Graybiel *et al.* (1944) in an analysis of 1000 healthy young aviators, could find no example of inversion of T in leads I or IV. However, Johnson (1940) during periodic

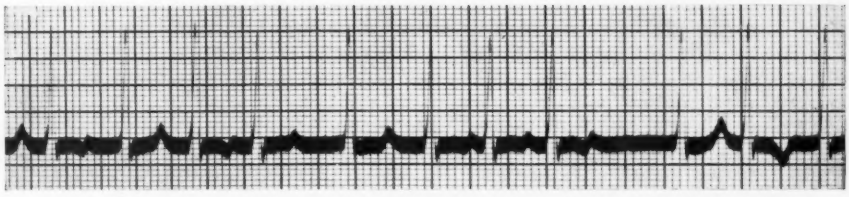


Fig. 11.—Case 10. Lead CF 4 recorded during an attack of cardiac pain. The T wave shows at times alternation, and varies from being sharply upright to being deeply inverted from beat to beat. No Q waves are seen. Auricular fibrillation is present.

routine examinations of 2400 apparently healthy males of average age 48 years, found that 1 per cent had an inverted or diphasic T 1 and T 2, but only standard leads were employed and the degree of inversion is not stated. Hall *et al.* (1942) using the three standard leads discovered among the cardiograms of 2000 members of the Royal Canadian Air Force who were fit for air crew, only two records (0·1 per cent) in which T 1 was negative and considered abnormal. In a study of 4810 soldiers, mostly psycho-neurotic, Stein (1946) could find only four (0·1 per cent) of them with inversion of T 1 and T 4 and in three of these the T inversion disappeared after exercise. Plooij (1946) states emphatically, as a result of his investigation of functional variations of the T wave, that T 1 is never inverted in health. The conclusions of Graybiel and White (1935) are similar. It would therefore seem reasonable to conclude that the T wave changes in our cases were all of pathological origin.

Considerable evidence has accumulated that reflexes originating elsewhere than in the heart, in particular in the alimentary tract, can cause cardiographic changes strongly reminiscent of cardiac infarction. It is well known that, clinically, disease of the gall bladder, peptic ulcer, and hiatus hernia of the stomach can all produce symptoms that simulate angina pectoris. It has been suggested that such diseases can be responsible *per se* for changes in the cardiogram that resemble those of myocardial infarction. Clarke (1945) has shewn that the cardiogram may return to normal after the cure of a duodenal ulcer or of cholecystitis, and Brown (1946) and Fitz-Hugh and Wolferth (1935) quote similar cases. Morrison and Swalm (1940) showed how, by dilating the œsophagus and stomach of a patient suffering from angina pectoris, changes in the cardiogram could be produced, but this included depression of the RS–T segment in lead 1. But the cardiographic changes as illustrated are minimal and could scarcely be confused with our records. In addition, to the best of our knowledge none of our patients had disease of the alimentary tract. The conclusion is that there is no reason on clinical grounds to suppose that the abnormal changes in our cases are due to derangement in the coronary circulation as a result of reflexes originating from lesions in the alimentary tract or elsewhere.

Of recent years the term " coronary insufficiency " is used to explain abnormality in the cardiogram that may be associated with cardiac pain. The pain can be induced by creating artificially a state of general cardiac anoxia, and the associated changes in the form of the cardiogram are used by Master (1946) and by Levy and his associates (1938, 1940, 1941, 1942) as an index of the functional efficiency of the heart. Levy insists on the importance of deviation of the RS–T segment. In a later paper, Patterson, Clarke, and Levy (1942) emphasized how the incidence of significant changes in the T wave increases with the degree of total RS–T deviation. The records so obtained do not resemble ours.

It would seem then that our records are not a normal variant, neither are they the result of reflexes arising from disease outside the heart, nor are they due to general cardiac anoxia. Some local abnormality in the myocardium, of a temporary and reversible nature, is likely to be present. Recently we have encountered two cases that throw some light on the problem.

The first patient, a woman aged 41, under the care of Dr. Clifford Hoyle, suffering from polyarteritis nodosa, complained of retrosternal pain during the night and her cardiogram next day showed that T 1, T 2 and T V4 were inverted and yet no Q waves were present (Fig. 12). At autopsy there was no infarct, but three small nodules were found at the tip of the front of the left ventricle. On microscopy these were confirmed to be due to polyarteritis nodosa, and many of the larger branches of the coronary arteries were also involved in that disease. The second case was a man, aged 56, with a blood pressure of 180/100, who had been suffering from very severe cardiac pain, both at rest and on exertion and only of three months' duration. The attacks of pain never lasted more than four minutes. His cardiogram, less than two days before he died, showed inversion of T 1, T 2 and T VL and T V2–6 inclusive, but with no Q waves present (Fig. 13). Yet at autopsy not only were his coronary arteries quite free from atheroma, but the muscle of the heart was quite normal to close naked eye scrutiny and to minute section of it. There was nothing pathological in the microscopical section, from the right and left ventricle and the septum.

It is possible therefore to have cardiac pain accompanied by cardiographic changes suggesting infarction, but without Q waves, where, in fact, local pathological changes other than infarction are present. Sometimes with these curves no naked eye disease whatsoever is present, and yet the patient may succumb. For many years the occasional absence of pathological changes in the myocardium in some patients who have died of angina pectoris has attracted the attention of clinicians, even to the extent of giving rise to Allbutt's theory that the pain was the " cry of a diseased aorta." The abnormal curves in this type of case show that the myocardium may be at fault even though no sign of disease can be detected at autopsy.

It has, of course, been known for twenty years that a cardiogram taken during and shortly after

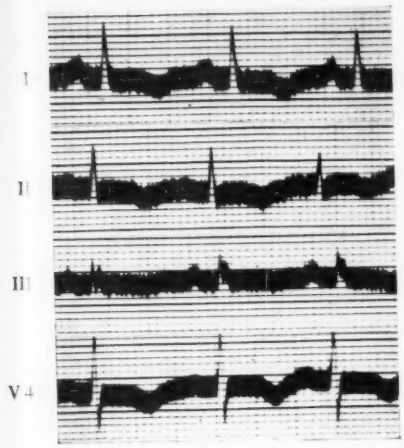


FIG. 12.—Cardiogram from a woman aged 41 who died of polyarteritis nodosa. At the front of the tip of the left ventricle there were several small nodules present which section revealed to be due to polyarteritis nodosa. No infarct was present.

anginal attacks, may show certain transient changes. Feil and Siegel (1928) presented such records in four cases where cardiac infarction could be excluded, and in three of these they recorded a depression of the RS–T segment in leads 1 and 2 during the pain and a return to normal after the pain had ceased. Shortly afterwards, Parkinson and Bedford (1931), employing standard leads, investigated five patients in none of whom was there any question of cardiac infarction at the time the records were taken, and in all five, during the pain the RS–T segment elevation or depression was noted. In four, the cardiogram had reverted to normal within forty minutes, and although the time taken for the fifth one to revert is not stated, it was normal by the time the attack of pain had ceased. On the other hand, Kahn (1931) studied the cardiograms of 330 cases of angina pectoris and observed that 8 of the 23 fatal cases died with no abnormality in the cardiogram, while they were suffering from repeated severe attacks. Emphasis on the characteristic RS–T segment shift was reiterated by Riseman *et al.* (1940). In a paper devoted to the study of 20 patients with angina, before, during, and after the attacks, they stated that the changes were most frequent and most striking in the præcordial leads, for in these, in all 20 cases, the RS–T segment was deviated. They were of the opinion that only later, towards the end of an attack and also after the disappearance of the pain, did the T wave changes become pronounced. Wilson and Johnson (1941) studied in detail cardiographic

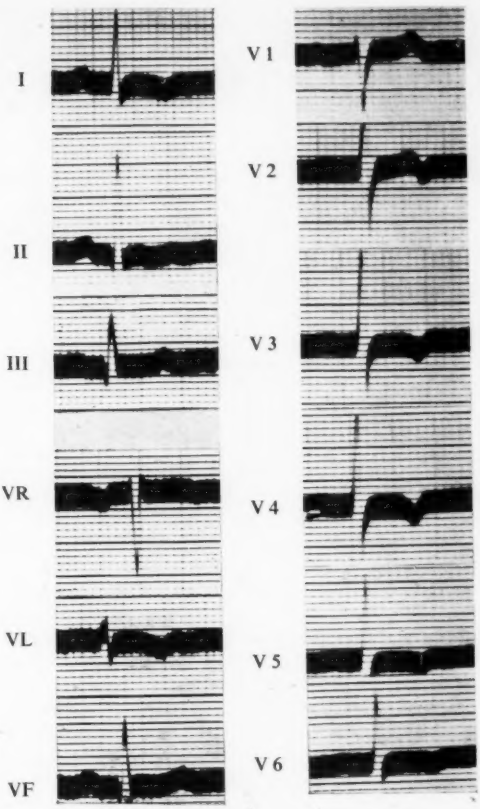


FIG. 13.—Record from a man aged 56 suffering from severe, but very short, attacks of cardiac pain. The cardiogram was taken less than two days before he suddenly died during a paroxysm of pain. At autopsy the heart and coronary vessels were quite normal, both to naked eye and microscopical examination.

changes obtained during the paroxysms of substernal distress, either spontaneous or induced by exercise or cigarette smoking, in five cases of angina pectoris, and in all five there was displacement of the RS–T segment.

It would seem then that the abnormal features shown by our cases are not simply those of the late stages of angina pectoris, as the segment shift was absent or negligible, the duration of the cardiographic changes was much longer, and many of our cases had prolonged pain at rest.

Coming nearer to infarction itself, Feil (1937) thought that he could distinguish a type of pain that precedes coronary thrombosis, but this pain

does not resemble that experienced by our patients and neither do the cardiograms he obtained whilst they had this pain resemble ours. Although Sampson and Eliaser (1937) also considered that it is possible to diagnose impending acute coronary occlusion, they gave no cardiographic details.

Once an infarct has occurred, provided that enough cardiac muscle has died, there is good experimental evidence, as produced by Wilson *et al.* (1933) in dogs, to show that a Q wave will be present. This suggests that in our cases not much muscle, if any, could have died. Certainly, in Case 10 of our series (Fig. 11) in which the T wave in CF 4 varied so much from beat to beat, it is difficult to see how this could result from other than temporary metabolic disturbances. Perhaps the clue to the lesion lies in the work of Bayley and his associates. As a result of experimental work on dogs, Bayley and La Due (1944) state that the electrical phenomena resulting from occlusion of the anterior descending branch of the left coronary artery are entirely independent of *demonstrable* pathological changes, and that the ischæmia injury changes, though often associated with infarction in man, are not to be regarded as necessarily diagnostic of infarction. In another paper, Bayley, La Due, and York (1944) used a unipolar lead in contact with the anterior surface of the pericardial sac and caused sudden complete occlusion of a dog's coronary artery, short of producing an infarct. They concluded that, contrary to the common belief, the first stage in the cardiographic evolution of myocardial infarct is the appearance of primary T wave changes which are ascribed to local ventricular ischæmia. In man, Bayley and Monte (1943) encountered a unique case. A hypertensive man experienced cardiac pain while at rest and was considered, by estimating the areas under curve QRS and under curve T and calculating Å QRS, Å T, and Å QRS–T, to show a severe degree of ischæmia in the distribution of the right coronary artery. This area diminished later and at autopsy the acute ischæmia was seen to have resulted from a dissecting aneurysm involving this artery. No cardiac lesion, naked eye or microscopic, was present. They concluded that " acute left ventricular ischæmia of sufficient intensity to produce striking T wave changes of a kind similar to those that occur in man with myocardial infarction, and lasting for days or even weeks, may be accompanied by no structural changes in the myocardium." They also call attention to the independent variation in magnitude and duration of acute local ischæmia, on the one hand, and pain on the other. It is not yet known how long local ischæmia, associated with only primary T wave changes, must exist in order to be followed by local myocardial fibrosis.

We conclude, therefore, that in our cases the presence of what we regard as primary T wave changes are the result of ischæmia of the myocardium caused by spasm of the coronary arteries. We have noted also the absence of displacement of the RS–T segment and the absence of Q waves. The reduction in the calibre of the vessel is not sufficient in degree or duration to lead to death of the myocardial fibres and subsequent fibrosis, or, if it does so, to anything but the slightest extent. The essential thing is coronary spasm; coronary atheroma is not a necessary feature. We do not know what causes such spasm nor why the left coronary artery should be involved so much more frequently than the right. Lisa and McPeak (1940) believe that coronary spasm without atheroma can lead to what they term " acute miliary infarction," and that infection is the precipitating cause of the spasm. Although their histological findings bear a striking resemblance to those seen in our Case 1, infection was not a feature of our cases, except possibly in Case 6.

Summary

When patients thought to be suffering from cardiac infarction were followed up, a small proportion of them were found in whom the cardiogram returned to normal and remained so. Most of these patients were hypertensive, and in more than half of them the attacks of pain ceased though the hypertension persisted.

If the cardiogram of such patients is compared with that of indisputable infarction, it will be found that almost all of them are anterior in distribution, that the Q waves are absent or insignificant, and that, with very few exceptions, there is no current of injury however early the record is taken. On the other hand, we do not claim that a patient yielding such a curve as we have described, but not returning to normal, has necessarily a good prognosis ; and we give an example where such a patient died suddenly, autopsy revealing no abnormality whatsoever. The term " recovery of the T wave " does not necessarily imply recovery of the patient, although the prognosis in the group as a whole is surprisingly good.

It is, of course, well known that the majority of patients who experience cardiac pain that comes on at rest and lasts for, say, more than half an hour, will have an abnormal cardiogram. But we would draw attention to the error of assuming that a patient with such pain, but with a normal cardiogram, cannot be suffering from cardiac pain. Several of our cases had pain that was at first, and on more than one occasion, accompanied by a normal

record, which only later showed the expected pathological changes. We suggest that prolonged spasm of the coronary artery is responsible for the cardiographic appearance, and only if the spasm is of sufficiently long duration does any fibrosis result, and this is minimal.

Since submitting this paper we have encountered six further cases; five were anterior and one posterior.

References

Bayley, R. H., and La Due, J. S. (1944). *Amer. Heart J.*, **28**, 54.
—, ——, and York, D. J. (1944). *Ibid.*, **27**, 657.
——, and Monte, L. A. (1943). *Ibid.*, **25**, 262.
Brown, H. (1946). *Clin. Proc.*, **5**, 447.
Clarke, N. E. (1945). *Amer. Heart J.*, **29**, 628.
Feil, H. (1937). *Amer. J. med. Sci.*, **193**, 42.
——, and Siegel, M. L. (1928). *Ibid.*, **175**, 255.
Fitz-Hugh, T., and Wolferth, C. C. (1935). *Ann. Surg.*, **101**, 478.
Graybiel, A., McFarland, R. A., Gates, D. C., and Webster, F. A. (1944). *Amer. Heart J.*, **27**, 524.
——, and White, P. D. (1935). *Ibid.*, **10**, 345.
Hall, G. E., Stewart, C. B., and Manning, G. W. (1942). *Canad. med. Ass. J.*, **46**, 226.
Hamburger, W. W., Katz, L. N., and Saphir, O. (1936). *J. Amer. med. Ass.*, **106**, 902.
Johnson, H. J. (1940). *Ibid.*, **114**, 561.
Kahn, M. H. (1931). *Ann. intern. Med.*, **4**, 1499.
Lenègre, J., and Chevalier, H. (1946). *Arch. Mal. Coeur*, **39**, 37.
Levy, R. L. (1938). *Amer. Heart J.*, **15**, 187.
Levy, R. L. (1941). *Bull. N.Y. Acad. Med.*, **17**, 898.
——, Bruenn, H. G., and Williams, N. E. (1940). *Amer. Heart J.*, **19**, 639.
Lisa J. R., and McPeak, E. (1940). *Arch. intern. Med.*, **65**, 919.
Master, A. M. (1946). *New York Med.*, **2**, 19.
Morrison, L. M., and Swalm, W. A. (1940). *J. Amer. med. Ass.*, **114**, 217.
Parkinson, J., and Bedford, D. E. (1931). *Lancet*, **1**, 15.
Patterson, J. E., Clark, T. W., and Levy, R. L. (1942). *Amer. Heart J.*, **23**, 837.
Plooij, M. (1946). *Arch. Mal. Coeur*, **39**, 126.
Riseman, J. E. F., Waller, J. V., and Brown, M. G. (1940). *Amer. Heart J.*, **19**, 683.
Sampson, J. J., and Eliaser, M. (1937). *Ibid.*, **13**, 675.
Stein, I. (1946). *J. Lab. clin. Med.*, **31**, 837.
Wilson, F. N., and Johnston, F. D. (1941). *Amer. Heart J.*, **22**, 64.
——, ——, Hill, I. G. W., and Grout, G. C. (1933). *Trans. Ass. Amer. Phys.*, **48**, 154.
White, P. D. (1945). *Heart Disease*, 3rd ed., New York, p. 207.

THE HEART IN THE PNEUMOCONIOSIS OF COALMINERS

BY

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Received September 6, 1948

The pneumoconiosis of coalminers is a primary disorder of the lungs that can cause pulmonary heart disease, a condition also called cor pulmonale.

The occurrence of right heart changes in pneumoconiosis was noted by Pancoast and Pendergrass (1931). Dyson (1933 and 1934) described the radiological recognition of heart disease in pneumoconiosis, and in a review of 127 cases found 18 with evidence of pulmonary heart disease. Discussing the pathology of pneumoconiosis, Jaffe (1934) concluded that the replacement of large parts of the lungs by poorly vascularized scar tissue, and the occurrence of obliterative changes in the branches of the pulmonary artery, interfered greatly with the pulmonary circulation. Right ventricular hypertrophy followed and failure of the right heart was a common cause of death in advanced pneumoconiosis.

Coggins *et al.* (1938) found that hypertrophy of the right ventricle was a common finding in the autopsy records of 102 proven cases of pneumoconiosis. Other studies of the pathology of pulmonary heart disease record right ventricular hypertrophy with anthraco-silicosis (Scott and Garvin, 1941; Spain and Hantdler, 1946). In the South Wales anthracite coalfield, Gooding (1946) has reviewed a series of 227 post-mortem examinations on miners certified as having pneumoconiosis; 37 per cent are described as having died from cardiac failure, usually right-sided.

Emphysema is a common cause of pulmonary heart failure. Gough (1940, 1946, 1947) has described the occurrence of emphysema in coalminers' pneumoconiosis and has differentiated the focal emphysema from the bullous emphysema that occurs in later stages of the disease. Geever (1947) has confirmed the occurrence of pulmonary vascular changes.

The clinical course of a pulmonary disease may be divided into two phases, the pulmonary phase and the cardiac phase (Brill, 1939). The clinical features of the cardiac phase in pneumoconiosis have been described by several writers, some of whom have indicated the difficulties in assessing the degree of involvement of the right heart (Coggins *et al.*, 1938; Giering and Charr, 1939; and Ketterer, 1941).

McMichael and Sharpey Schafer (1944) have drawn attention to the fact that cases of cor pulmonale may have a high cardiac output. They have found this in patients with emphysema, gross venous congestion, and a low arterial oxygen saturation. In the terminal stages the left heart may fail and pulmonary œdema occur.

The pneumoconiosis of coalminers has been presented in detail by Fletcher (1948).

Selection of Cases

The subjects of this investigation have been inpatients under the care of the Pneumoconiosis Research Unit (M.R.C.) at Llandough Hospital. A certain measure of selection has been exercised in this series because of the exclusion of patients with any signs of cardiovascular disease other than that resulting from the pulmonary disorder. There are 96 cases of coalminers' pneumoconiosis ranging from the earliest detectable stage to the final stage of advanced disease and right heart failure. Their ages vary from 28 to 65 years, but the severity of the disease is not proportional to the age. For ease of description a clinical grading has been adopted, based on the history, the degree of dyspnœa, and the capability for exertion.

The Clinical Examination

The symptoms of the pulmonary phase were dyspnœa, fatigue, cough, and intermittent attacks of pain in the chest. The dyspnœa was exertional and slight at first, but usually increasing in severity. Paroxysmal dyspnœa was not uncommon and was not cardiac in origin. A complaint of being easily fatigued was often an early symptom. Cough was present at some stage, sometimes productive in type. The pain in the chest was the result of the pulmonary

TABLE I

CLASSIFICATION OF CASES

		No. of cases
Grade I	Those not definitely disabled	14
Grade II	Those with slight disability	35
Grade III	Those with moderate disability	15
Grade IV	Those with serious or complete disability	32

disease although sometimes simulating pain of cardiac origin. The site of the pain in the chest varied from time to time, and its onset was not always related to exertion. The physical signs of the pulmonary phase were similar to those of emphysema and chronic bronchitis without heart disease.

The symptoms of the cardiac phase were severe dyspnœa, gross disability, œdema of the feet and ankles, and upper abdominal discomfort. The patients would often complain only of marked exacerbation of their previous condition.

The physical signs of the cardiac phase were orthopnœa, cyanosis, engorgement of the neck veins, warm hands, œdema of the ankles, tenderness of the palpable liver, a full peripheral pulse of regular rhythm, and triple heart rhythm.

Orthopnœa might be the earliest sign of imminent heart failure and might be related to the history of an exacerbation of the pulmonary phase. Cyanosis was occasionally present with the advanced pulmonary phase but was often a sign of right heart failure. Engorgement of the neck veins was present only when the heart failed; it has not been seen in this series as a result of the pulmonary disorder alone. Warm hands were noted when failure occurred but the sign was less distinctive as failure persisted. Œdema of the feet or ankles appeared as a very early finding and quickly increased to a severe degree. The liver might be palpable in the pulmonary phase owing to the low diaphragm but tenderness was limited to the cardiac phase. The pulse was full and usually of regular rhythm. The blood pressure in all these cases was between 100 and 150 mm. systolic, and 50 and 90 mm. diastolic. The majority had a blood pressure of the order of 110 systolic and 70 diastolic. The heart size and position were difficult to assess. The cardiac impulse was felt in the epigastrium. The heart sounds were distant except at the sternal margins and in the epigastrium. Triple heart rhythm (protodiastolic gallop) was heard to the left of the lower sternum in 75 per cent of the cases when the right heart failed. There was accentuation or splitting of the second sound in the pulmonary area. A systolic murmur of moderate intensity was heard in the mitral area in two cases. Slight sinus tachycardia was evident at complete rest and the rate accelerated with the slightest exertion. One patient was exceptional in having auricular flutter; there were no records of auricular fibrillation. The hæmoglobin levels were within normal limits except in two cases where the readings were below normal. Severe jaundice was noted in one case of severe failure and milder jaundice in another.

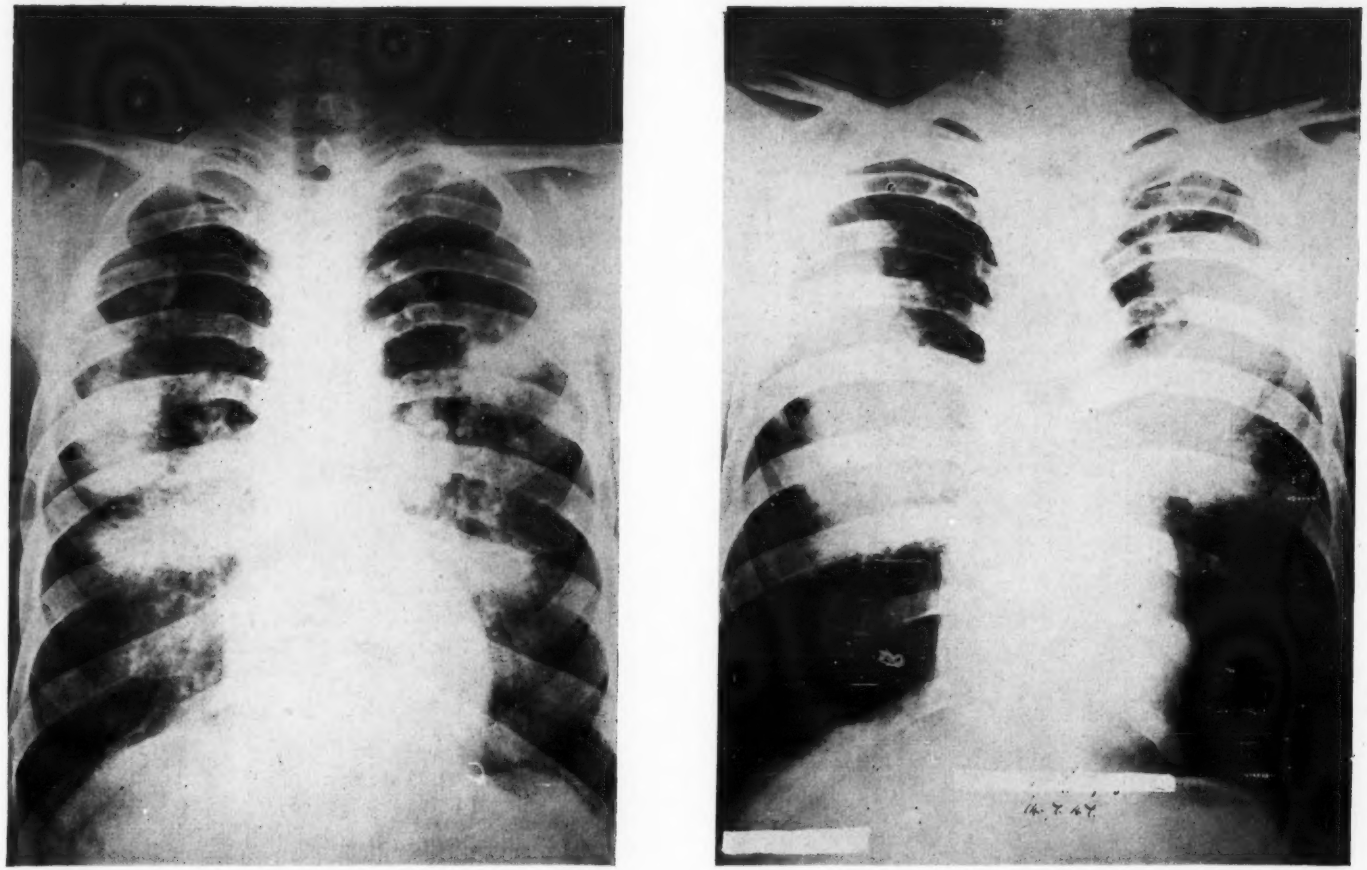
Right heart failure was present in 11 cases: in 9 of them it was fatal.

The stage at which right heart involvement first occurred was difficult to detect. One half of those in the pulmonary phase showed a maximal impulse at the lower end of the sternum and had accentuation or splitting of the pulmonary second sound. The splitting of the second sound was often audible down the left border of the sternum. In one instance a short diastolic murmur was audible at the pulmonary area with the breath held in inspiration. The signs found in the pulmonary area can be classed as suggestive of right heart strain. The clinical examination is not very helpful in detecting early right heart involvement.

THE RADIOLOGICAL EXAMINATION

The clinical worth of radiology in the study of heart disease has been stressed by Parkinson (1936). The pathological studies of Kirch (1930) on the right ventricle and its inflow and outflow tracts are applicable to this work. Kerley (1931), Schwedell (1946), and many others have described the radioscopic features of right ventricular enlargement.

Enlargement of the right ventricle takes place first in the outflow tract and is seen as a prominence of the pulmonary conus–artery segment. There is straightening of the upper left cardiac border in the anterior view and an anterior bulge reducing the retrosternal space in the right anterior oblique view. Right ventricular inflow tract enlargement is manifested by increasing width and depth of the right ventricle in the anterior and the left anterior oblique views, particularly in the latter. Tomography of the



A B

FIG. 1.—(A) July 1941. (B) The same patient in July 1947, aged 45 years. The heart shape has changed. The right ventricular outflow tract is enlarged in 1947 (Fig. 4).

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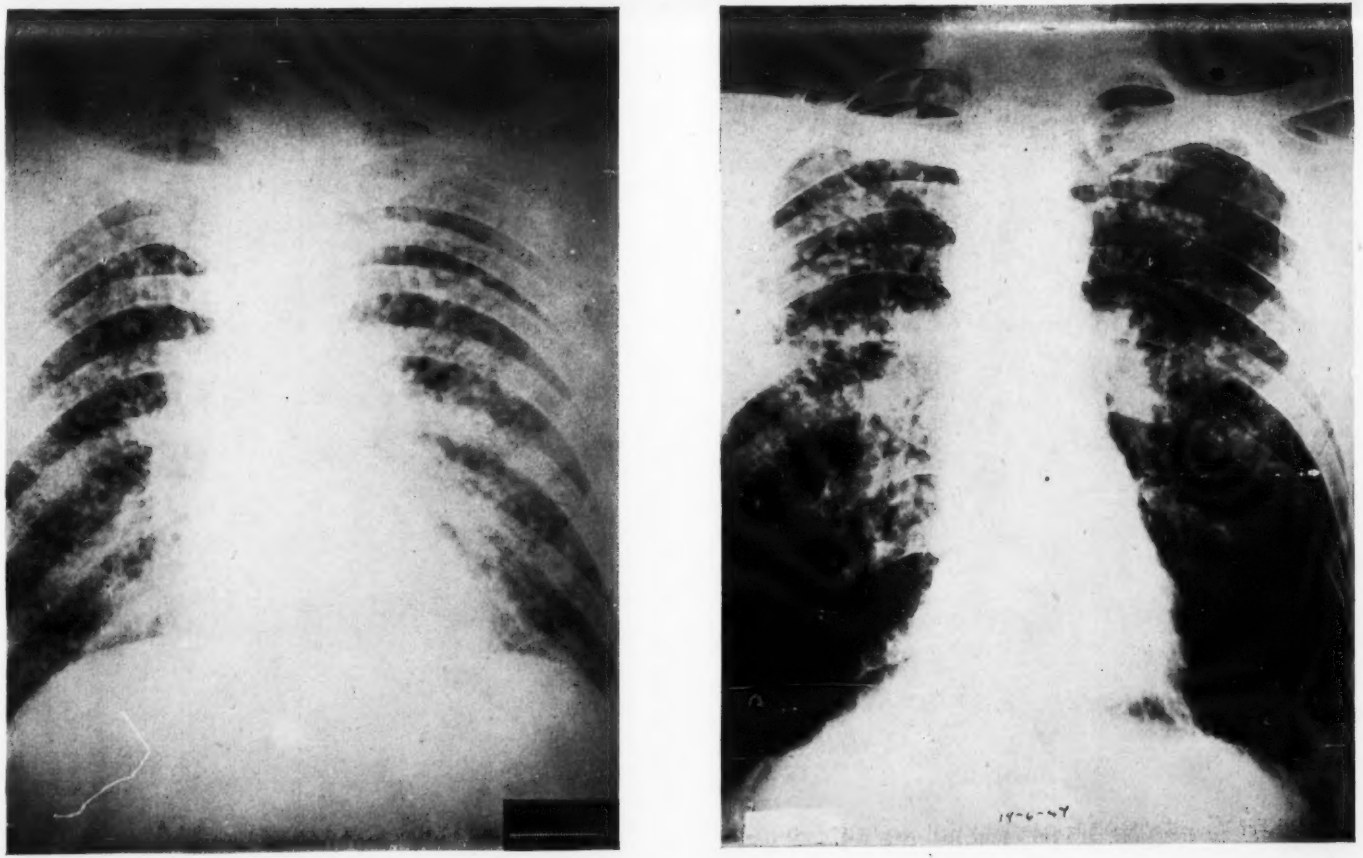

A B

FIG. 2.—(A) October 1938. (B) The same patient in June 1947, aged 55 years. The left cardiac border has lengthened and now shows the prominence of the pulmonary artery and below the artery the pulmonary "conus."

heart shadow in the right anterior oblique view has been found of value in recording enlargement of the right ventricular outflow tract.

Two noteworthy changes occur in the shape of the heart in pneumoconiosis; one is elongation and assumption of a vertical position, the other is enlargement of the outflow tract of the right ventricle.

The general heart shape is analysed in Table II. In ten instances, where previous records are available, analysis proves that marked elongation of the heart shape has occurred; radioscopy now shows that the original heart shape cannot be reformed by any respiratory movement (Fig. 1 and 2). The diaphragm has been depressed to produce the elongation, and in some a further real or apparent elongation is produced by the elevation of the lung hila (see Fig. 5).

Five patients in right heart failure had heart shadows rather wider than is expected (Fig. 3). The belief that widening has occurred due to right ventricular inflow tract enlargement is supported by the demonstration of this enlargement in two cases on radioscopy, and by the measuring of actual widening of the shadow in successive teleradiograms of a third case. Even so, the radiological and pathological size of the hearts of those in failure is small, relative to those with the same degree of heart failure from other cardiovascular diseases.

Enlargement of the pulmonary artery and of the outflow tract is a finding of a major importance. Their detection is not easy when much elongation is present and is even more difficult when there is gross distortion. The incidence of this finding is given in Table II.

Further changes may not be detected in some cases for many years. During this interval the heart usually elongates vertically. The final stage is reached when the inflow tract dilates and right heart failure follows very quickly.

The period of prominence of the outflow tract only is presumably that of increase in pulmonary artery pressure and may be called the stage of right heart strain.

The Electrocardiographic Examination

The electrocardiographic pattern of right ventricular hypertrophy has been described by Wilson *et al.* (1930, 1944, 1947) and Myers *et al.*, (1948). The three standard leads are affected by the position of the heart and the examination of præcordial lead tracings is essential in studying the right heart. The presence of right axis deviation in the standard leads accompanied by inversion of T II and T III is not diagnostic of right ventricular hypertrophy, (Myers *et al.*, 1948). In pronounced right ventricular hypertrophy abnormally large R waves with a late peak, frequently Q waves and inverted T waves, are found in leads from the right side of the præcordium. In leads from the left side of the præcordium R is often smaller than usual and an S wave persists. The QRS deflections are not usually large, nor is the QRS interval wider than normal.

The standard limb leads have been recorded in all cases in this series. An analysis of the axis deviation is shown in Table III.

The common pattern in the standard lead tracings is that associated with the long vertical heart—QRS of small amplitude in lead I, R wave of good amplitude in leads II and III, and frequently a small Q wave in leads II and III. Inversion of the T wave in leads II and III occurs in records from 8 patients, 6 of whom were in clinical grade IV.

A high P wave in lead II has been described in pulmonary heart disease by Winternitz (1935) and others, but there is still doubt as to its true significance. Thirty-two records show a P wave in lead II of an amplitude between 2 and 3 mm.; fourteen

TABLE II

Radiological Changes in the Heart

Clinical grades	No. of cases	Long vertical heart shape	Right ventricular outflow tract enlargement
I	14	1	Nil
II	35	8	2
III	15	7	9
IV	32	21	24

Prominence of the pulmonary conus-artery segment has been detected in 4 cases without elongation of the heart shape or heart failure. The remaining 31 with enlargement of the outflow tract include 11 cases that had signs of right heart failure (Fig. 3, 4, and 6).

Pulmonary conus-artery enlargement has taken place at a comparatively early stage in 2 cases, and has been found with moderate disability in 9 others.

B

A

FIG. 3.—(A) Anterior view. (B) Lateral view. From a patient, aged 47. In the lateral view the anterior prominence and high curvature of the enlarged right ventricle is seen. This patient was in right heart failure. The right ventricular hypertrophy was confirmed post-mortem. (See also Fig. 7B.)

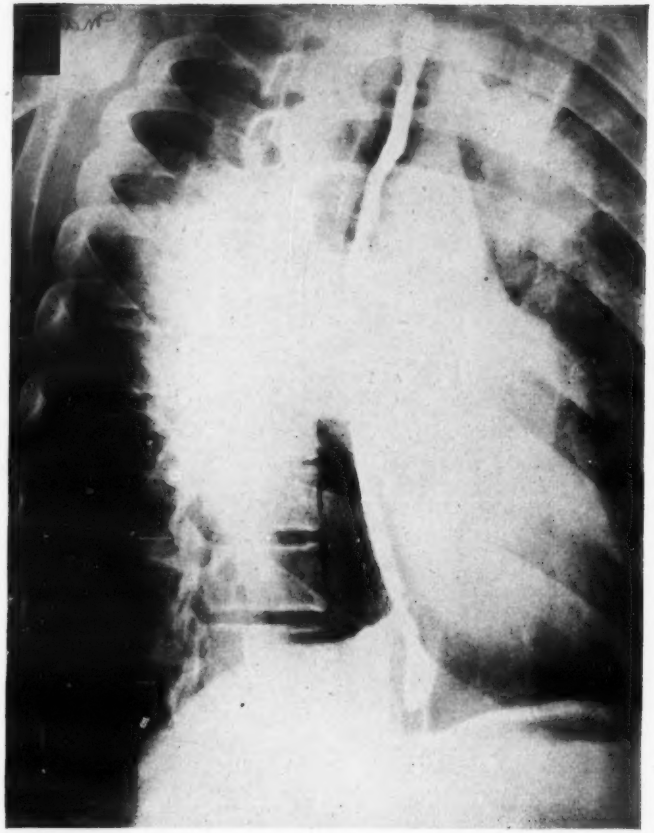

FIG. 4

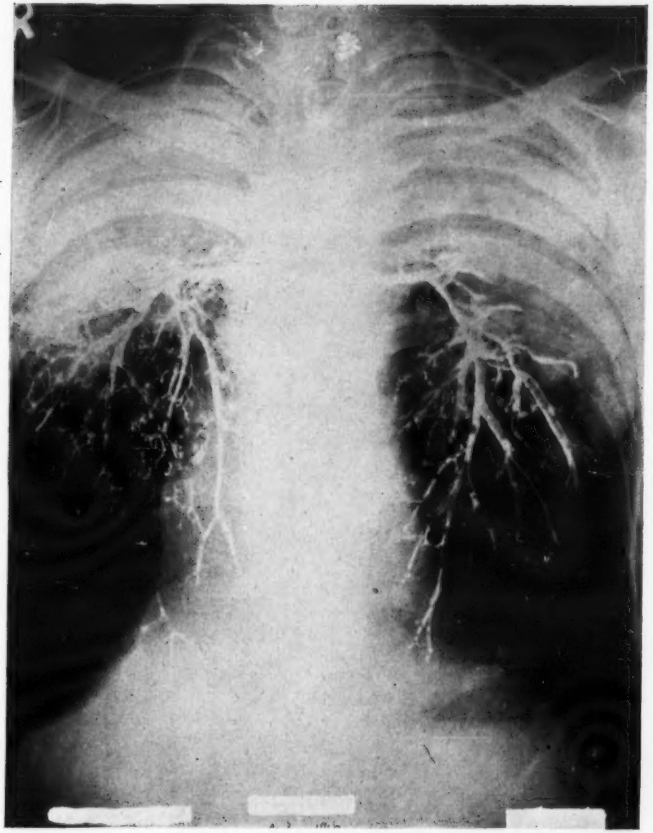

FIG. 5

FIG. 4.—Right anterior oblique view of the heart shown in Fig. 1B. Enlargement of the outflow tract of the right ventricle is seen as a prominence of the upper portion of the anterior border.

FIG. 5.—Bronchogram, May 1947. This shows the effect on the heart shape of the distortion, depression of the diaphragm, and elevation of the hila.

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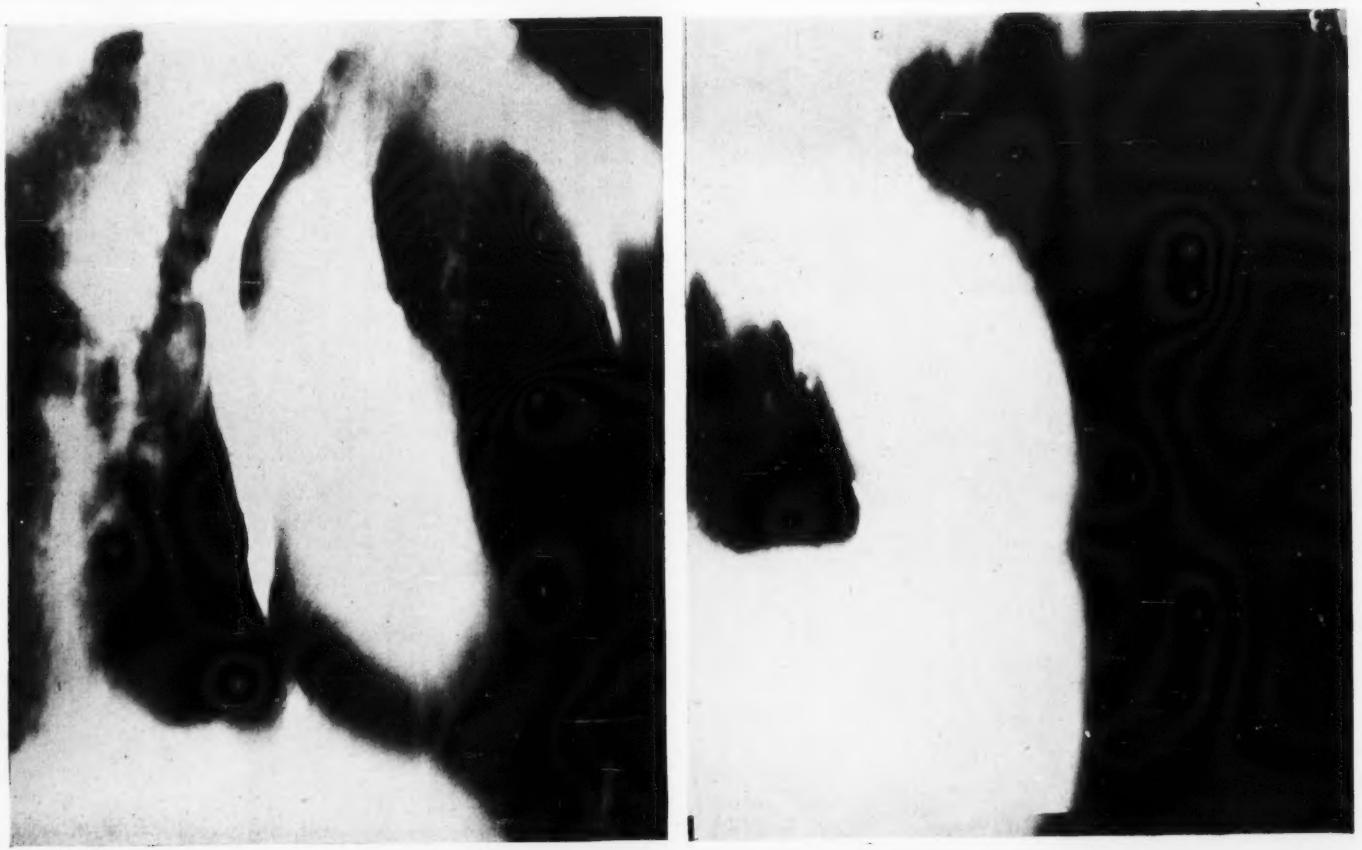

A B

FIG. 6.—(A) and (B) Tomograms of two patients taken in the right anterior oblique view to record the enlargement of the right ventricular outflow tract, which had been seen on radioscopy.

TABLE III

AXIS DEVIATION IN ELECTROCARDIOGRAMS

Axis	0° to + 29°	+ 30° to + 59°	+ 66° to + 89°	+ 90° to + 120°	Over + 120°
Cases	12	13	40	28	3

have a P wave over 3 mm. amplitude in lead II. The P wave is abnormally high in these fourteen cases, ten of whom are in clinical grade IV. It only coincides with other detectable signs of right heart involvement in nine cases. The possibility that rotation of the heart is responsible for the high P wave of lead II cannot be excluded (Fig. 7).

The unipolar limb leads have been recorded in twenty cases. VL has a QS deflection in fifteen, which is indicative of the vertical heart position. VR in five tracings has an R wave suggestive of right ventricular hypertrophy.

Chest leads have been recorded in all cases, CR leads in the first 30 and V leads in the other 66 cases. In eight tracings the pattern of pronounced right ventricular hypertrophy was seen (Fig. 7). All had radiological evidence of right ventricular prominence and post-mortem confirmation of the hypertrophy of the right ventricle has been obtained in three of them.

There is a second group of eight tracings with a distinctive pattern. The QRS complex in leads from the right side (V4R, V3R, V1) is of low amplitude and has a RSR′ form; leads from the left side of the præcordium show a persistent S wave. This pattern is suggestive, but not evidence, of right ventricular hypertrophy (Fig. 7D). Three of these patients had right heart failure and the right heart hypertrophy has been confirmed post-mortem; three others had radiological evidence of right ventricular prominence.

A third group of ten tracings shows a predominantly S wave pattern from the right side of the chest with a small S wave persisting through V4 to V6 (Fig. 7E). The R wave is of normal character in the leads from the left chest. This pattern is not evidence of right ventricular hypertrophy but is of interest because six of these cases were considered to have right heart strain and the right ventricular hypertrophy has been confirmed at autopsy in two of them. It is apparent that rotation has played a part in producing this pattern and the taking of leads from other positions may be of value in confirming the change in the heart.

Twenty-six records thus show abnormality but the classical pattern of ventricular right preponderance is found only in eight. The second chest lead pattern described above deserves to be classed as suggestive of right heart involvement. It is clear that the position of the heart, changed as it may often be in this disease, can modify the chest lead pattern and it is important therefore to take leads from positions further to the right than lead V1 and from as far left as V6.

In thirty cases unipolar leads from the right upper abdominal quadrant and from the left upper scapular region have been taken (Goldberger, 1944). The results were not conclusive and heart position again seemed to cause variation. The majority had an upright complex in the abdominal lead, and a downward complex in the scapular lead; a few were diphasic in nature and of very low amplitude.

CONCLUSIONS

Right heart failure occurs in the late stage of pneumoconiosis of coalminers as a result of the pulmonary lesions. When this happens, the clinical picture is the same as that found in heart failure due to other pulmonary diseases; it is true pulmonary heart disease. There is radiological evidence of the right ventricular enlargement and in some cases there is electrocardiographic evidence of right ventricular hypertrophy. Other factors such as rotation of the heart or distortion may mask the typical cardiographic or radiological features.

In the early stages of pneumoconiosis, no evidence of right heart involvement can be detected by the methods used in this investigation.

There are cases in the intermediate stages in whom evidence of right heart strain has been found. Accentuation and splitting of the pulmonary second sound are early signs suggestive of an increase in the tension in the pulmonary artery. Later in the disease it has been possible to demonstrate a prominence of the pulmonary artery and of the outflow tract of the right ventricle. The electrocardiograph has not been as helpful in this group, but has sometimes provided supporting evidence of right ventricular hypertrophy. This right heart strain can persist for a long period, amounting to some years, before the final stage of right heart failure appears. Clearly not more than 50 per cent of the total will develop right heart failure.

The order and frequency of appearance of the signs of right heart strain are only to be expected

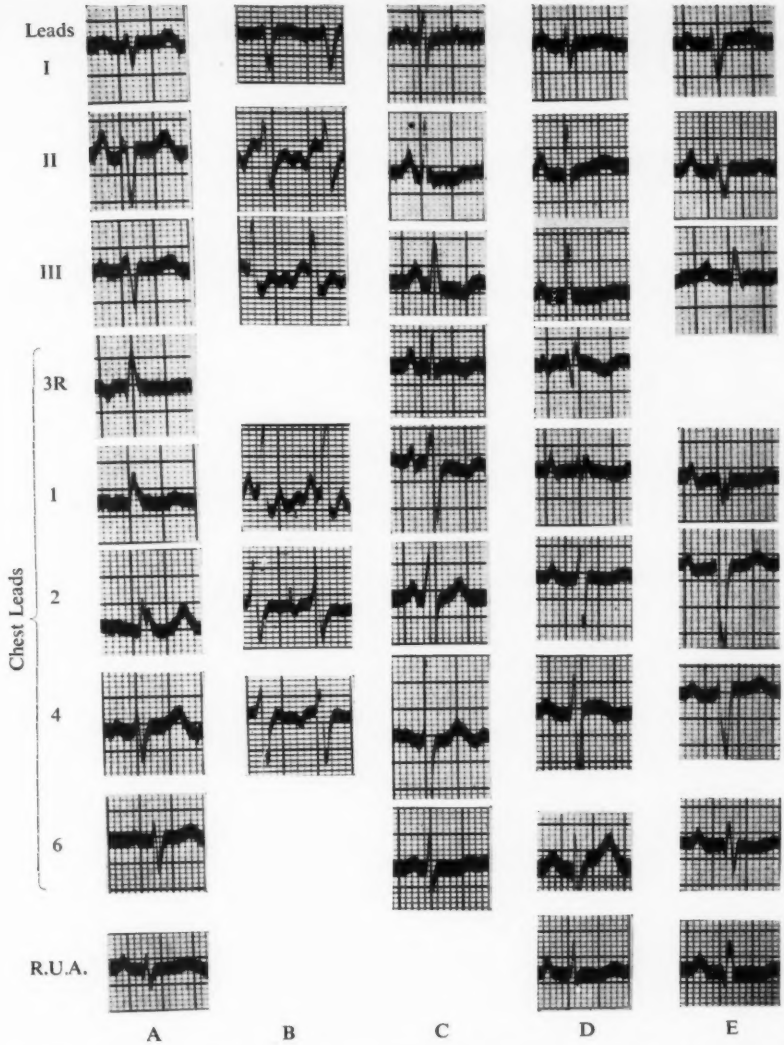

FIG. 7.—Cardiograms of five patients with right heart involvement. The chest leads in (B) are CR leads, all others are V leads.

(A) Clinical grade IV. The pattern is that of right ventricular hypertrophy. This agreed with the other findings.

(B) The pattern is that of right ventricular hypertrophy but auricular flutter is also present. The same case as in Fig. 3.

(C) Right ventricular hypertrophy which was confirmed later, post-mortem.

(D) Clinical and radiological evidence of right heart strain; the pattern is placed in the second group described in the text as suggestive of right heart strain.

(E) The "S" wave chest lead pattern placed in group three in the text, and not taken as evidence of right heart strain. It is probably caused by rotation of the heart. The presence of right ventricular hypertrophy was confirmed later in this case.

R.U.A. is the unipolar lead taken from the right upper abdominal quadrant.

when it is realized that the initial causation is the change in the dynamics of the pulmonary circulation. Increase in the pulmonary arterial pressure first affects the pulmonary second sound, then dynamic enlargement of the pulmonary artery appears. There follows a dynamic enlargement of the right ventricular outflow tract and it is only when hypertrophy takes place in this tract that the electrocardiographic changes can be expected. The progression is slow but super-added respiratory infections can precipitate the process.

The relating of the signs of right ventricular strain to the pulmonary changes is difficult. Cournand (1947) has drawn attention to the presence of pulmonary hypertension in patients with pulmonary fibrosis and moderate emphysema, when pulmonary hypertension has not been found in those with moderate emphysema alone. Right heart strain is present in cases in this series without severe bullous emphysema but, as shown by Gough, focal emphysema, fine radiating fibrosis, and pulmonary vascular changes may be present. The clinical disability of such cases is only moderate in degree, but the findings in this investigation suggest that pulmonary hypertension is already present. A closer investigation of the respiratory functions of patients with early right ventricular strain may help to elucidate the factors involved in the causation of pulmonary hypertension.

Summary

A clinical study of the cardiovascular system has been made in 96 patients with pneumoconiosis of coalminers. Eleven patients had right heart failure and another twenty-four had evidence of enlargement of the right ventricular outflow tract. Radiological examination has been of most value in detecting early right heart involvement. Electrocardiographic examination has been helpful in some cases, and the cardiographic patterns are described.

I wish to thank Dr. C. M. Fletcher, Director of the Pneumoconiosis Research Unit (M.R.C.) for permission to study the cases under his care and for access to the records of these cases. A part of this work was incorporated in a Thesis accepted by the University of Wales in 1947.

References

Brill, I. C. (1939). *Ann. intern. Med.*, **13**, 513.
Coggin, C. B., Griggs, D. E., and Stilson, W. L. (1938). *Amer. Heart J.*, **16**, 411.
Cournand, A. (1947). *Bull. N. Y. Acad. Med.*, **23**, 48.
Dyson, J. M. (1933). *Amer. J. med. Sci.*, **186**, 165.
—— (1934). *Amer. Heart J.*, **9**, 764.
Fletcher, C. M. (1948). *Brit. med. J.*, **1**, 1015, 1065.
Geever, E. F. (1947). *Amer. J. med. Sci.*, **214**, 292.
Giering, J. F., and Charr, R. (1939). *J. Amer. med. Ass.*, **113**, 574.
Goldberger, E. (1944). *Amer. Heart J.*, **28**, 621.
Gooding, C. G. (1946). *Lancet*, **2**, 891.
Gough, J. (1940). *J. Path. Bact.*, **51**, 277.
—— (1946). *Lancet*, **1**, 462.
—— (1947). *Occup. Med.*, **4**, 86.
Jaffe, R. H. (1934). *Illinois med. J.*, **66**, 431.
Kerley, P. (1931). *Recent Advances in Radiology*, London.
Ketterer, C. H. (1941). *Med. Bull. Veterans' Adm.*, **18**, 98.
Kirch, E. (1930). *Klin. Wschr. Berl.*, **9**, 769.
McMichael, J., and Sharpey Schafer, E. P. (1944). *Quart. J. Med.*, **13**, 123.
Myers, G. B., Klein, H. A., and Stofer, B. E. (1948). *Amer. Heart J.*, **35**, 1.
Pancoast, H. K., and Pendergrass, E. P. (1931). *Amer. J. Roentgenol.*, **26**, 556.
Parkinson, J. (1936). *Lancet*, **1**, 1337, 1391.
Schwedell, J. B. (1946). *Clinical Roentgenology of the Heart*, p. 103, *Ann. Roentgenol.*, New York.
Scott, R. W., and Garvin, C. F. (1941). *Amer. Heart J.*, **22**, 56.
Spain, D. M., and Handler, B. J. (1946). *Arch. intern. Med.*, **77**, 37.
Wilson, F. N., Macleod A. G., and Barker P. S. (1930). *Amer. Heart J.*, **6**, 637.
——, ——, Johnston, F. D., and Barker, P. S. (1933). *Ibid.*, **9**, 447.
——, *et al.* (1944). *Ibid.*, **27**, 19.
——, Rosenbaun, F. F., and Johnston, F. D. (1947). *Advances in Internal Medicine*, **2**, 37.
Winternitz, M. (1935). *Med. Klin.*, **31**, 1575.

PROCEEDINGS OF THE BRITISH CARDIAC SOCIETY

The TWELTH ANNUAL GENERAL MEETING of the British Cardiac Society was held at the Liverpool Medical Institution on Thursday, May 13, 1948. Chairman: John Hay. The Chairman took the chair at 9.30 a.m.; 82 members and 12 visitors were present.

PRIVATE BUSINESS

1. The minutes of the last Annual Meeting, having been published in the Journal (**9**, 304, 1947) were taken as read and confirmed.
2. The balance sheet for 1947–48 was presented, having been audited by Hay and Wyn Jones. The credit balance on April 21, 1948, was £88 13*s*. 4*d*.
3. The meeting confirmed the action of the Council (under Minute 7 of the last meeting) in conferring Honorary Membership on:
   Dr. Helen Taussig, Baltimore
   Dr. Alfred Blalock, Baltimore
   Dr. Harold Pardee, New York
   Dr. Andre Cournand, New York
   Dr. Camille Lian, Paris
4. Cotton was elected as a member of the Council in place of Wallace-Jones (resigned). Cookson and I. G. W. Hill were elected in place of Peel and Parsons-Smith (terms of office expired).
5. Athelstone Hill was elected as an *Extra-Ordinary Member*.
6. The following Associate Members were elected as *Ordinary Members*:
   Arnott, Lendrum, Hunter, O'Donovan, Kerley.
7. The following new *Associate Members* were elected:
   G. Aitken, Glasgow
   C. G. Baker, London
   R. E. Bonham Carter, London
   E. M. Buzzard, Oxford
   W. E. Clarke, London
   D. H. Davies, Bristol
   Frances Gardner, London
   J. Gibson Graham, Glasgow
   T. E. Gumpert, Sheffield
   G. Hayward, London
   S. Oram, London
8. The following *Associate Members* were elected for a further period of three years:

R. J. Duthie	E. Graham Jones
J. G. M. Hamilton	B. B. Morgan
F. M. Hilliard	W. R. Snodgrass
J. C. Hoyle	A. J. Wilson

9. The following addition was made to Rule 6:
   "In addition there may be 10 Ordinary Members who may be elected for their interest in cardiovascular surgery."
   It was agreed that the Council should decide from time to time whether Rule 12 should be enforced as regards these members.
   After discussion it was agreed that surgeons specializing in surgery of the sympathetic should be included under this addition to Rule 6 and it was also agreed that the operation of this rule should not hinder election of new Ordinary Members from the list of Associate Members.
10. The following Surgeons were elected as Ordinary Members under the new addition to Rule 6:

R. C. Brock	C. Price Thomas
P. R. Allison	O. S. Tubbs
T. Holmes Sellors	Vernon C. Thompson

11. Resignations were received and accepted from:
    A. G. Biggam, I. de Burgh Daly, M. Kremer.
12. After discussion it was agreed that an Autumn Meeting should be held this year as an experiment, some time in October, and that the place of the Meeting should be left to the Council for decision.

DISCUSSION ON SYMPATHECTOMY IN HYPERTENSION

Opened by ROBERT PLATT, RAE GILCHRIST, CLIFFORD WILSON, and W. T. COOKE

ROBERT PLATT (*introduced*). Although sympathectomy may be described as dangerous, unphysiological, and undesirable, it must be admitted that in a certain number of cases—probably 30 to 40 per cent—it achieves results that are clearly worth while and cannot be achieved by any other method in use at the present time. In some of these the blood pressure is brought from dangerous levels into a range of comparative or complete safety. It is not proposed to discuss how sympathectomy acts but it is important to be sure that the patients are better for the reduction in blood pressure. Undoubtedly they are, for symptoms disappear, the heart size diminishes, electrocardiograms improve, and retinopathy may disappear, and there is no evidence of deterioration in renal function as a result of the operation.

The question of selection is difficult and makes it

usually impossible to compare one series with another. If the procedure were harmless and reliable obviously it should be used in an early stage of the disease. As it is neither, the speaker has reserved operation for cases in which the prognosis is otherwise judged to be bad. These are usually patients under 50 years of age with diastolic pressures remaining at 125 or more after adequate rest, provided that renal and cardiac function are not badly affected, or arteriosclerotic changes advanced. Early signs of impending cardiac failure or a transient cerebral incident have been regarded as indications for operation as soon as possible. Such a selection is hard on the surgeon and liable to increase the mortality.

Pentothal and other depressor tests show a general correlation with the results of sympathectomy in that the patients who do not respond to the one rarely respond to the other, but there are exceptions and a poor response to depressor tests has not been regarded as ruling out treatment by operation.

In Manchester an extensive operation has been done by Boyd in two, three, and sometimes four stages, the sympathetic chain being removed from the 4th dorsal to the 3rd lumbar, since G. A. G. Mitchell (*Edin. med. J.*, 1947, **54**, 545) has shown that this is necessary in order to be reasonably certain that the sphlanchnic area is denervated.

Table I shows the results to date in 54 cases. These are not classified with regard to ætiology but successes have included essential hypertension, chronic pyelonephritis, and hypertension resulting from pregnancy kidney. The cases described as of malignant type have all shown papillœdema with very high diastolic pressures.

TABLE I

RESULTS OF SYMPATHECTOMY, MAY 1948

	Total	Malignant type
Still incomplete or too recent ..	10	4
Failed to complete operation ..	4	3
Deaths	10	3
Failures after complete operation	14	3
Worth while result	5	3
Good result	11	1
Total	54	17

Table II shows the change in diastolic pressure after operation, and it will be noted that in the 11 cases classified as " good results " the average post-operative blood pressure is at a very satisfactory level. Cases recorded as " worth while " would appear to have had their pressures reduced to much safer (though still not satisfactory) levels.

TABLE II

SYMPATHECTOMY CASES, MAY 1948

Group	Average age	Average pre-op. pressure	Average post-op. pressure (lying)	Change in diastolic
Good results ..	36	210/130	140/90	— 40
Worth while results ..	38	230/145	180/110	— 35
Failed	37	230/140	230/140	0

A. H. Radcliffe, in Professor Boyd's department, has been using a sensitive method of charting electrical skin resistance and finds that in the failures the area of skin which has been efficiently sympathectomized is much smaller than in the successful cases. The correlation is not exact but the investigation suggests that in many cases failure is due to the fact that efficient sympathectomy has not been achieved. This may be due to anatomical anomalies.

Owing to a number of deaths having occurred as a result of the operation a physician was detailed to watch some of the operations. The sudden falls of pressure which may occur as the result of anæsthesia, movement of the patient, and the operation itself, are extremely alarming, and it is felt that some of the fatalities could have been avoided if the anæsthetists had had a clearer appreciation of the dangers. A sudden fall in pressure can usually be combated by methedrine.

RAE GILCHRIST. As the leading cause of death and with complications more widespread and more devastating than the ravages of malignant disease, the degenerative process labelled " hypertension " remains our greatest medical problem. The pressing need at the present time is the development of methods for the prevention of vascular damage at the sites that take the brunt of the burden, namely the retinal, renal, cerebral, and coronary systems. Until such time as a rational specific therapy is available, as in diabetes or pernicious anæmia, treatment necessarily falls short of the ideal and symptomatic measures, whether medical or surgical, must be employed in a palliative sense. We recognize their inadequacy.

It might well be thought that the evaluation of a precise method of treatment such as sympathetic surgery offers for a state of affairs so common and so easily recognized as essential hypertension should not present any great difficulty. The reverse, however, is the case. An adequate knowledge of the

course and complications of hypertension is unfortunately lacking. In consequence it is difficult to compare the results obtained by medical and surgical methods. There are so many interacting factors such as age, sex, duration, severity, renal function, retinal grouping, and, above all, vascular vulnerability, that it is difficult to present for study exactly comparable groups—the one treated surgically and the other medically. Recognizing the limitations and admitting the crude method of analysis employed, a comparison of the results obtained, based on a study of 126 cases of hypertension, has led to the following tentative conclusions. Our data have not yet been submitted to specialized statistical methods and are therefore open to criticism. Our results are largely impressions.

Eighty patients suffering from benign hypertension received medical care exclusively and forty-six received in addition surgical attention. All were subjected to detailed investigations according to a predetermined routine and have been followed for periods up to 5 and 8½ years, respectively.

Symptoms as a test of cure are misleading in that their intensity cannot be gauged with accuracy, and the spontaneous fluctuations and natural remissions, which are a feature of the disease process, make accurate deductions all the more difficult. Nevertheless, in the medical group symptomatic improvement was observed, though the changes were relatively slight. In the surgical group, in whom symptoms were distinctly more severe, symptomatic improvement was striking. For instance, in the medical group, at the end of 3 years, 20 per cent were symptom free, and in the surgical group approximately 50 per cent. We do not have sufficient facts to warrant the conclusion that sympathectomy postpones the date of death from vascular disease.

Progress has also been assessed on the response of the diastolic blood pressure. This sign is also admittedly fallacious, for if there is one finding more misleading than another, more liable to unpredictable fluctuations, it is the blood pressure level. The diastolic pressure has a smaller range than the systolic and we have therefore attempted to make use of it for comparative purposes by recording the figures in the horizontal position after at least half-an-hour's rest, the subject being thoroughly accustomed to the surroundings, the technique, and the observer. In the medical group the tendency is for the diastolic pressure to rise with the passage of time. Comparing the findings at the end of 3 years, there is an increase in the number of patients with a diastolic pressure over 120, just as there is a decrease in the number of those with a diastolic pressure 100 or less. There is therefore a tendency for the diastolic pressure to rise, despite a reduction in the severity of the symptoms. In the surgical group at the end of 3 years there is an increase in the number of patients with diastolic pressures under 100 and some reduction in the number of those in the middle ranges, without much alteration in the proportion of patients with diastolic pressures over 120. It therefore appears that 3 years after sympathectomy a significant fall of diastolic pressure remains evident in approximately 20 per cent of the patients submitted to surgery. There were 5 fatal cases in the 46 submitted to surgery. All died from vascular causes.

Thirty patients suffering from malignant hypertension have been under observation, 10 of whom had sympathectomies done—a number too few to justify statistical analysis, but the facts observed are worth recording. The 20 patients treated medically without exception died within six months of coming under observation. Of the 10 receiving surgical treatment, 3 obtained no benefit and in the light of further experience would not now be submitted to surgery. Moderate benefit, enabling a return to work for a year or two, in 2 patients was encouraging. The remaining 5 have made remarkable improvement, all showing a regression in the retinitis and a subsidence of the papillœdema, without, however, any significant change in the level of the diastolic pressure. Our most remarkable case is a man aged 47, well and in steady employment 4 years after sympathectomy. He came under observation on account of hæmoptysis with papillœdema.

It is in this group that sympathetic surgery can claim its greatest achievements. Provided renal function is reasonably good and heart failure does not threaten, a bilateral sympathectomy can arrest the degenerative process, restore vision, relieve symptoms, and apparently prolong life.

Surgery is not the ideal treatment of hypertension. As a palliative measure it is capable of relieving symptoms, even when these are severe, in about 50 per cent of patients, carefully chosen for this procedure. It has little effect on the diastolic blood pressure level: in only about 20 per cent is the diastolic pressure restored to normal. These results are far from ideal, but they are better than those obtained by purely medical means.

The contra-indications to surgical intervention are arteriosclerosis, angina, congestive failure, and impaired renal function. Minor cerebral episodes, even transient hemiplegias, do not necessarily contra-indicate surgery. In the desperate condition labelled "malignant hypertension" surgery can postpone the fatal outcome, restore vision, relieve symptoms, and enable the sufferer to return to work—an achievement that has hitherto not resulted from any medical measures so far employed.

CLIFFORD WILSON (*introduced*) presented the results

of lumbo-dorsal sympathectomy in 46 cases of hypertension followed for six months to five years. Twenty-five cases were diagnosed malignant hypertension, eleven severe benign hypertension in young subjects, and ten primary renal disease. The effect of cerebral and cardiac complications, and of renal impairment on the surgical risk was discussed. In general the malignant hypertension syndrome carried a greater risk than any of these complications taken singly but cardiac asthma or renal failure with blood urea up to 100 mg. usually contra-indicated operation.

Analysis of results showed that during the five-year period 18 of the 46 cases had died; 14 of these were diagnosed malignant hypertension. Mortality was greater and survival periods shorter in males than in females. Examining the diagnostic groups separately: in *malignant hypertension* where the need for some form of therapy is most urgent, the operative risks are grave and the results disappointing. In selected cases the operation may increase the expectation of life by two or three years. In the present series, the longest survival period was 4¾ years; in almost all cases there was symptomatic relief particularly from headaches, and retinal changes, including papillœdema, improved or disappeared. *Severe benign hypertension* in young subjects would seem to provide the most favourable indication for sympathectomy but unfortunately improvement is largely subjective and the ultimate effect on prognosis is difficult to assess. In our group of 10 cases there were no deaths, but sustained fall in blood pressure was observed in only 3 patients.

In *primary renal disease* (bilateral) with severe hypertension but with good renal function, the results of operation were encouraging and it seems possible that in these patients progressive renal deterioration may be retarded.

In conclusion, more emphasis should be placed on diagnosis; renal biopsy should be performed at operation in order to confirm the clinical diagnosis. In benign hypertension the natural history of the disease should be more carefully studied so as to provide a control group against which the operative results would be more reliably assessed.

W. T. Cooke. In Birmingham, a Smithwick operation has been performed upon 50 patients more than 12 months ago by Mr. W. H. Sweet, Brodie Hughes, and J. M. Small. The main features are summarized in Table III.

In the absence of a large control group, blood pressure changes and survival rates are of little value in assessing the merits of the operation. The procedure appears to have been beneficial for certain subjective reasons—the disappearance of severe headaches and lassitude, and increased capacity for work. Thus of the whole group, approximately 25 per cent, including 5 of Grade IV severity, are working full time in factory or the home, approximately one-third are dead or invalids, whilst the remainder are improved and not prevented from working, though still incapacitated to some extent. Objectively 2 out of 30 of the patients showed decrease in heart size, the remainder no change. Hæmorrhages and papillœdema cleared up in all those in whom they were present. Of 25 patients with abnormal cardiograms, 10 became normal after operation and 4 showed only left axis deviation.

In 25 cases, renal plasma flow and glomerular filtration rate was determined, and in 20 cases, effective kidney mass. These determinations though revealing all stages of kidney impairment, gave no indication as to whether an operation might be successful. Renal biopsy on 16 patients also revealed the great diversity of kidney pathology, which was

TABLE III

	Grade I and II	Grade III	Grade IV	Total
Men	5	3	11	19
Women	16	6	9	31
Average age	42	41·8	40	41 (10–58)
Average blood pressure	236/143	233/137	240/144	237/142
Average fall	47/25	70/35	43/34	
Normal blood pressure	—	2	2	4
Deaths	1	0	8	9
Untraced	2	—	2	4
Average survival	Living (18) 30 months	Living (9) 31 months	Living (10) 35·8 months	

Average survival whole group living and dead	28·7 months
Average time since operation	32·5 months

often completely unsuspected clinically. For these reasons, it is of utmost importance that renal biopsies should be taken as a routine if any accurate assessment of results is to be obtained in the future.

Cerebral catastrophes which had occurred in 15 patients did not prove a contra-indication. Six are working full time. Four have normal blood pressures including two who have each two normal pregnancies. Four are dead. Of four patients with congestive cardiac failure, one responded dramatically and is now working.

In this series, there were patients in whom the operation appeared life-saving; and others to indicate that there was a place for surgery in the treatment of hypertension and papillœdema below the age of 50; for progressive hypertension in the young age groups with signs of incipient failure of cerebrum, kidneys, or heart; or at any age for the relief of the intractable type of headache occurring in hypertension. Contra-indications are advanced kidney failure, generalized arteriosclerosis, recent cerebral hæmorrhage, or any case with marked functional overlay.

Short Communications

Measurement of Pulse Volume

By E. B. Colegrave and T. G. Richards
(*introduced*)

A method is described by which true pulse volume may be measured at any external pressure. A triple armlet is used and volume is directly measured by a liquid lens of negligible inertia.

It was shown that the orthodox graph of pulse volume against external pressure cannot give a derived curve of arterial collapse. The instability in collapse which makes this impossible is found to be related to the total arterial rigidity, which may then be calculated. The internal pressure valves are given by subtracting the valve of internal rigidity from the clinical blood pressure figures.

Arterial resistances to collapse in normal subjects are found to be of the same order. Much higher figures are found in arteriosclerosis. In true hypertension the figure depends upon the severity and duration of the disease. Arterial rigidity may occur before electrocardiographic changes are evident.

Complete Heart Block Associated with Amœbic Hepatitis

M. D. Rawkins and G. L. S. Konstam
(*introduced*)

An ex-army officer, aged 28, complained of fainting attacks over a period of three days and pain over the lower sternum aggravated by breathing. In 1942 whilst serving in the Western Desert he had periodic attacks of mild diarrhœa, for which he did not receive treatment, and since then occasional diarrhœa, but none for nine months.

There was no history of rheumatic fever nor diphtheria but he had had scarlet fever, when 19, and since then occasional pains in the limbs.

On examination (1/1/48) he was well nourished. Resting pulse 40 and regular. B.P. 145/70. Heart not enlarged, no murmurs. Liver dullness extended upwards in the mid-axillary line to fourth space, edge not felt.

Cardiogram showed C.H.B. and X-ray examination showed that the right dome of the diaphragm was considerably raised, immobile, and its outline blurred. The transverse diameter of the heart was slightly increased to the left and right. The appearances were typical of amœbic hepatitis but stool examinations were negative for *E. histolytica* and sigmoidoscopy showed normal appearances.

A course of eleven emetine HCl (gr. 1) injections was started and after the third injection the pulse rate increased to 80, with a P–R interval of 0·2 sec., the chest pain disappeared, and the Stokes-Adams attacks ceased.

A further X-ray examination showed free movement of the diaphragm and the right dome had returned to its normal level and was regular in outline.

Heart block associated with amœbiasis is a rarity. We have found in the last twenty-three years three other cases of A-V dissociation. (Petzetakis, *Arch. Mal. Coeur*, 1925, **18**, 70; Gerbasi, *La Pediatria.*, 1931, **39**, 513; Heilig and Visveswar, *Indian Med. Gaz.*, 1943, **78**, 419.) In each this complication occurred during active dysentery and response to emetine seemed certain in only one.

In the course of prolonged diarrhœa other factors that could influence the conducting tissue are anæmia, toxæmia, thiamin and nicotinic acid deficiency, peripheral circulatory failure, uræmia, and possibly abnormal blood and tissue chemistry.

The case here described had not had diarrhœa for nine months and both the hepatitis and heart block appeared to respond promptly to emetine. An amœbic metastasis in the heart therefore seemed more probable than an indirect toxic effect on the conducting tissues.

Stokes-Adams Attack in Latent Heart Block

By G. Bourne

Two cases of Stokes-Adams attacks were described in which the underlying disease of the bundle was not suspected to be cause of the attacks; one had been examined by a cardiologist and the other by a general physician. In each the history of the attacks aroused suspicion as to their possible cause. The first patient was a man of 70 who for seven months previously at intervals of a month or less had had constant attacks of syncope. These were abrupt in onset, lasted three to five seconds, and were not followed by any headache, drowsiness, or any other sequelæ. He was just as well after the attacks as before. The heart showed moderate enlargement on cardioscopy. There was a harsh systolic murmur at the apex base. The heart rate was 56 and was regular. The blood pressure was 116/72. The cardiogram showed a P–R interval of 0·2 sec. and a prolonged QRS complex. He was given ephedrine 0·5 grains t.i.d., and from that day, a period of 21 months, has remained free from attacks.

The second patient was a woman of 61 with a two years history of from four to eight weeks interval between attacks, but the frequency of the attacks had increased for a month or two previous to examination. The attacks were abrupt and were followed by no sequelæ, nor were they preceded by any warning. They lasted ten seconds, for the husband was in the habit of counting ten seconds by which time he knew that his wife would recover. She had been thoroughly investigated for epilepsy. There was an aortic systolic murmur. The blood pressure was 150/90. The cardiogram showed an increased P–R interval of 0·23 sec. Cardioscopy showed moderate enlargement. Further tracings taken to exclude a previous infarct were done and in two of these a true heart block was shown. The purpose of showing the cases was to stress the importance of an exact history in suspecting examples of Stokes-Adams attack in patients hitherto unsuspected of heart block.

Heart Block in Osteitis Deformans

By C. V. Harrison and B. Lennox (*introduced*)

The authors reported two cases. The first was a woman of 71 who, while in hospital with advanced Paget's disease, developed Stokes-Adams attacks with temporary complete heart block. At autopsy there was calcification along the base of the mitral valve which had spread on to the interventricular septum to involve the bundle of His. The second was a man of 74 with advanced Paget's disease who was admitted with recent dyspnœa and found to have complete heart block. He died soon after admission and at autopsy there was calcification along the bases of the mitral and aortic valves and spread on to the septum involving the bundle of His.

The authors believe that this association between Paget's disease and cardiac calcification is not fortuitous. An analysis of 30 published post-mortem records on cases of Paget's disease revealed that 11 of them had cardiac calcification. Similarly, 13 cases of Paget's disease in their own departmental records included 6 cases with cardiac calcification.

From a comparison of these figures with those from a control series of routine autopsies, the authors concluded that cardiac calcification was five times as common in Paget's disease as in controls and that the heart block observed clinically in their two cases is to be regarded as a true complication of Paget's disease.

Two Newer Murmurs in Diastole

By William Evans

The first was a trivial or incidental murmur in early diastole, exocardial in origin, and found in cases of sternal depression; the murmur was not evidence of aortic incompetence.

The second was a continuous murmur in systole and diastole which is the outcome of a small and unimportant A-V fistula and should be distinguished from the murmur of patent ductus arteriosus in order to save patients from unnecessary operation. The phonocardiogram will decide both clinical problems.

Phonocardiography in Heart Disease

By E. D. H. Cowen (*introduced*)

A phonocardiographic investigation of systolic murmurs (1) in valvular and congenital heart disease (63 cases) and (2) in subjects without other evidence of cardiac disability (40 cases), revealed no significant difference in time of onset of the murmur in relation to the S line of lead II of the electrocardiogram in the two groups. This analysis was undertaken following the work of W. Evans (*Brit. Heart J.*, **9**, 1 and 225, 1947).

A new phonocardiograph evolved by the Cambridge Instrument Company in collaboration with the author was used, consisting of a piezo-electric crystal microphone, andion tube amplifier, and double fibre string galvanometer. A frequency response curve of this instrument was shown in a discussion on the calibration of phonocardiography.

Phonocardiograms taken by the new instrument were shown, in which it was demonstrated, confirming many previous workers, that there is no exact correspondence between the mechanical and electrical events of the cardiac cycle.

Angiocardiography as a Diagnostic Aid

By Frances Gardner

The angiocardiographic work described in this report is confined to congenital heart disease. The technique employed is that described by Robb and Steinberg in 1938 with the addition of a preliminary intravenous sensitivity test.

The film cassettes are changed by hand and only one exposure every three seconds is possible. The arm-lung circulation time is a reliable guide to the exposure time for the right heart. The arm-tongue circulation time is extremely unreliable as a guide to similar times for the left heart and aorta. Good contrast films of these structures are therefore difficult to obtain unless large quantities of radio-opaque material are used.

The slides demonstrate the angiocardiographic appearances in the following conditions.

1. Congenital dilatation of the pulmonary artery with stenosis and incompetence of the pulmonary valve.
2. Isolated pulmonary stenosis.
3. Patent ductus arteriosus.
4. Fallot's tetralogy.
5. Pulmonary atresia with patent ductus arteriosus.
6. Coarctation of the aorta.

There are probably few congenital heart lesions where angiocardiography is essential for accurate diagnosis. It is, however, valuable as a method of visualizing the precise anatomy of the pulmonary circulation and the site and extent of aortic coarctation.

A Clinical Comparison of CR, CF, and Unipolar Chest Leads

By Aubrey Leatham (*introduced*)

Leaving aside theoretical considerations of the relative advantages of certain chest leads now in common use, an investigation has been made of the clinical value of these leads. Standard limb leads, unipolar limb leads, and chest leads CR, CF, and V1 to 7 have been taken in over 300 cases so far, and the following conclusions have been reached.

In rare cases of cardiac pain the CF lead has shown changes in the T wave that were not found in either CR or V leads. Since such an event is so rare, and since the lead commonly shows similar changes in healthy subjects, and must then be read in conjunction with the unipolar limb leads, the practice of recording CF as the only chest lead should be condemned.

In CR leads, T wave positivity tends to be exaggerated which is an advantage in health, but in very rare cases may conceal small changes in the T wave that are indicative of disease; however, only once did the V chest leads show a change in a patient with cardiac pain that was not present in the CR leads.

A disadvantage of V leads is the fact that in health the T wave in V1 is often inverted, and the T waves in V6 and V7 are customarily low and may be flat, or even inverted in V7.

Tuberculous Pericarditis

By A. A. F. Peel

Published in full, Brit. Heart J., **10**, 195, 1948.

Cardiac Infarction Complicated by Bundle Branch Block

By W. Somerville (*introduced*)

An attempt has been made to estimate the frequency with which an electrocardiographic diagnosis of cardiac infarction can be made in the presence of bundle branch block.

A series of 58 cases each with a clear history of cardiac infarction and a cardiogram showing bundle branch block was examined. The cases fell into two groups; in one, the pathological signs of infarction were present in the cardiogram in addition to those of bundle branch block (37 cases); in the other group, the signs of infarction were suppressed (21 cases). There were 34 cases of left bundle branch block, in 15 of which the signs of infarction were present, and 24 cases of right bundle branch block, in 22 of which the signs of infarction were present.

The distribution of the abnormal Q and T waves, and RS–T segment deviations, diagnostic of cardiac infarction is set down in Table IV. The term chest leads refers to unipolar leads V1 to V6 or V7; in a few cases, only V1, V3, and V5 were taken. Unipolar limb leads refers to the Wilson leads VL, VR, and VF.

TABLE IV

Pathological Signs of Cardiac Infarction in Presence of Bundle Branch Block.

	Abnormal Q	*RS–T deviation*	*Abnormal T*
Right Bundle Branch Block			
Anterior infarction (11 cases)			
Limb leads (11 cases) ..	6/11	2/11	1/11
Chest leads (9 cases) ..	9/9	8/9	7/9
Unipolar limb leads (6 cases)	4/6	2/6	0/6
Posterior infarction (11 cases)			
Limb leads (11 cases) ..	10/11	10/11	9/11
Chest leads (8 cases) ..	1/8	0/8	1/8
Unipolar limb leads (2 cases)	2/2	1/2	1/2

LEFT BUNDLE BRANCH BLOCK

	Abnormal Q	*RS–T deviation*	*Abnormal T*
Anterior infarction (11 cases)			
Limb leads (11 cases) ..	10/11	2/11	11/11
Chest leads (9 cases) ..	7/9	7/9	6/9
Unipolar limb leads (5 cases)	4/5	1/5	4/5
Posterior infarction (4 cases)			
Limb leads (4 cases) ..	4/4	4/4	4/4
Chest leads (4 cases) ..	1/4	2/4	1/4
Unipolar limb leads (2 cases)	2/2	1/2	2/2

When dealing with such restricted numbers, it is not possible to draw firm conclusions regarding the frequency with which a diagnosis of cardiac infarction and bundle branch block can be made from the electrocardiogram. The findings in the present series indicate that when right bundle branch block complicates cardiac infarction, signs of infarction may be expected to be found in the majority (22 of 24 cases, or 92 per cent).

When left bundle branch block complicates cardiac infarction, the signs of infarction may be found in about half the cases (15 of 34, or 45 per cent). Emphasis should be placed on the fact that there is approximately an even chance that the electrocardiographic signs of a cardiac infarct will be suppressed if left bundle branch block is a complication.

ABSTRACTS OF CARDIOLOGY

Therapy Directed at the Somatic Component of Cardiac Pain. S. H. RINZLER and J. TRAVELL. *Amer. Heart J.* **35**, 248–268, Feb., 1948.

Observations were made on 31 patients with the symptoms of angina pectoris who presented "trigger areas" in the muscles of the præcordium. When the trigger areas were either infiltrated with procaine (0·25 to 0·5%) or sprayed with ethyl chloride relief of pain lasting for days was experienced by those patients whose angina had followed a coronary thrombosis, but not by those without such a history. *H. E. Holling*

Electrocardiogram in Chronic Cor Pulmonale. R. ZUCKERMANN, E. CABRERA, B. L. FISHLEDER, and D. SODI-PALLARES. *Amer. Heart J.* **35**, 421–437, March, 1948.

The authors are convinced that the electrocardiogram is a real aid in the diagnosis of chronic cor pulmonale and that often a diagnosis of this condition can be made by the electrocardiogram before it can be arrived at by clinical methods. They describe their electrocardiographic findings in detail and list 10 principal diagnostic signs. *R. T. Grant*

Septal Anastomoses. C. LAUBRY, P. SOULIÉ, and H. THYS. *Arch. Mal. Cœur*, **41**, 1–24, Jan.–Feb., 1948.

A study of the coronary artery anastomoses in the interventricular septum was made in 50 hearts by injecting "lipiodol" at low pressure (50 mm. Hg or less) into the coronary arteries, the septum being isolated. The injected specimens were examined radiographically. The authors conclude that: (1) in normal hearts, specially in those from young subjects, septal anastomoses are so frequent as to be considered the rule; (2) anastomoses are rare in elderly subjects. *R. T. Grant*

Complete Transposition of the Aorta and the Pulmonary Artery. Experimental Observations on Venous Shunts as Corrective Procedures. C. R. HANLON and A. BLALOCK. *Ann. Surg.*, **127**, 385–397, March, 1948.

Transposition of the aorta and pulmonary artery is a rare deformity, in the presence of which survival is only possible through the persistence of fœtal passages such as ventricular or auricular septal defects, patent ductus arteriosus, or entry of pulmonary veins into the right auricle. Length of survival depends on the amount of mixing of pulmonary and systemic blood that can take place. The average in 123 reported cases is 19 months, but one patient lived a normal life for 56 years.

Experiments were performed on dogs to discover whether venous shunts would permit survival in the presence of aortic and pulmonary artery transposition. The shunts were performed first, and consisted of anastomosis of the veins from the two upper lobes of the right lung into either the right auricle or the superior vena cava. The latter gave the better results, as there was less disparity in thickness between the vessels anastomosed. These shunts were shown to be patent some months after operation but in no case in which experimental transposition was subsequently effected has survival been possible for more than a few minutes. This is presumably because the shunt allowed insufficient mixing. Further attempts are being made. *J. B. Kinmonth*

The Determination of the Prognosis of Pregnancy in Rheumatic Heart Disease. J. J. BUNIM and J. RUBRICIUS. *Amer. Heart J.* **35**, 282–297, Feb., 1948.

Observations were made on 142 pregnant women with rheumatic heart disease through pregnancy and the puerperium; the literature on the subject was studied. It is concluded that pregnancy itself has little effect on the prognosis in rheumatic heart disease. *H. E. Holling*

Effect of Exercise on Cardiac Output and Pulmonary Arterial Pressure in Normal Persons and in Patients with Cardiovascular Disease and Pulmonary Emphysema. H. B. HICKAM and W. H. CARGILL. *J. clin. Invest.*, **27**, 10–23, Jan., 1948.

The changes in the circulation of 28 persons (8 normal subjects, 8 with congestive failure, 7 with mitral stenosis, and 5 with emphysema) were studied by means of cardiac catheterization at rest and during exercises in a supine position in which the patients pushed with their feet against weighted pedals. In the controls cardiac output and the arterio-venous oxygen difference both increased, though the increase in cardiac output was the greater. In congestive heart failure there was little change in cardiac output but a large increase in arterio-venous oxygen difference. The normal pulmonary vascular bed can accommodate the amount of blood associated with a large increase in the rate of blood flow, with little or no increase in mean pulmonary arterial pressure. With left ventricular failure mean pulmonary arterial pressure, already elevated at rest, rises much further on exercise. Similar changes were noted in well-marked mitral stenosis, but in some patients the increase appeared larger than could be accounted for by a fixed obstruction at the mitral orifice. In advanced pulmonary emphysema the elevated pulmonary arterial pressures noted at rest and on exercise were believed to result from destruction of small vessels in the lung. *W. T. Cooke*

Treatment of Thromboangiitis Obliterans and Thrombophlebitis with Tetraethylammonium Chloride. R. Buxton and E. C. Eickhoff. *Sth. Med. Surg.* **110**, 69–71, March, 1948.

The action of tetra-ethyl-ammonium chloride in establishing a temporary sympathetic block was used in the treatment of 6 cases of thrombo-angiitis obliterans and of 5 cases of thrombophlebitis, 2 to 3 ml. being injected up to 3 times daily for varying periods of time [the actual dosage is not given]. Among the cases of Buerger's disease, 1 is included in which the condition followed a fracture of the first lumbar vertebra. This patient and 2 others of this series were markedly improved by the treatment, although a lumbar sympathectomy preceded improvement in 1 of them. In 3 other cases treatment failed. In the 5 cases of thrombophlebitis pain ceased within 2 days of starting treatment, and the average time taken to effect a cure was 7 days. *G. Schoenewald*

The Effect of Tetra-ethyl-ammonium Chloride on Blood Pressure Before and After Sympathectomy for Hypertension. H. S. Brown, E. V. Allen, and W. McK. Craig. *Proc. Mayo Clin.*, **23**, 94–99, Feb. 18, 1948.

Tetra-ethyl-ammonium chloride blocks the transmission of impulses from preganglionic to postganglionic fibre of the autonomic nervous system. The effect is transient and includes a fall in blood pressure of hypertensive patients in the recumbent position, orthostatic hypotension, tachycardia, mydriasis, and increase in the skin temperature of the toes. In the present observations the drug has been found to reduce greatly the blood pressure of recumbent hypertensive patients who have undergone sympathectomy by the Smithwick operation. Compared with the pre-operative action of the drug, 25 to 30% of the dose has post-operatively two or three times the effect. Observations on 6 subjects are recorded. The significance of the effect is considered. The authors discard the possibility that it may be due to the drugs' acting in more concentrated manner on the relatively few remaining effective ganglia. They suggest that the fact that the residual post-sympathectomy hypertension can be reduced to normal indicates that organic changes in the arterioles are not an important factor in the persistent hypertension. *C. L. Cope*

Pathology and Clinical Features of Idiopathic Isolated Myocarditis. Y. G. Etinger and N. L. Vilk. *Klin. Med., Mosk.*, **26**, No. 3, 3–13, 1948.

The authors have been able to study 7 cases of idiopathic myocarditis both clinically and at necropsy, and consider that the condition may be diagnosed during the patient's life. The ætiology is obscure. Heart failure came on suddenly, sometimes after such conditions as physical exertion, pregnancy, or acute infection (influenza). In 4 cases the aorta was hypoplastic. Five patients were females; ages ranged from 20 to 60 years. The clinical picture was one of severe and rapidly progressive congestive heart failure with cyanosis and dyspnoea. The heart was enlarged; arrhythmias were common. The blood pressure was low, the pulse soft. Occasional bouts of pyrexia occurred. The electrocardiogram pointed to a diffuse lesion; voltage was low. Diagnosis was made by exclusion in 5 cases during life. At necropsy, coagulative necrosis and myolysis of the myocardium were sometimes the chief features, while in other cases exudative and proliferative changes predominated. *S. S. B. Gilder*

The Diagnostic Value of Electrocardiographic Patterns, Based on an Assay of 261 Additional Autopsied Cases. L. N. Katz, D. Feldman, and R. Langendorf. *Acta cardiol., Brux.*, **2**, 291–317, 1947.

This study is a continuation of previous work on the same subject. It is based on a series of 261 consecutive cases in which electrocardiograms (including chest leads) were made within 2 months of necropsy. The authors conclude that in general the electrocardiogram is a good index of whether or not the heart is structurally normal. An abnormal electrocardiogram is excellent evidence that the heart is abnormal. On the other hand, a normal electrocardiogram is occasionally found with an abnormal heart. In 41 cases of the series a discrepancy existed between the electrocardiographic diagnosis and the necropsy findings; these cases are discussed and separately tabulated. *R. T. Grant*

Studies of Fluorocardiography in Normal Subjects. F. G. Fleischner, F. J. Romano, and A. A. Luisada. *Proc. Soc. exp. Biol., N.Y.*, **67**, 535–539, April, 1948.

Fluorocardiography (electrokymography) is a newly developed procedure for recording on a continuous film the pulsations of various areas of the cardiovascular silhouette and the opacity changes of the lung fields on X-ray examination. Simultaneous phonocardiographic records are taken, which permit exact timing of the fluoroscopic pulsations. This paper is a further contribution to the study of fluorocardiography in normal subjects. Records of tracings of the ascending aorta, pulmonary veins, and pulmonary parenchyma are presented and their significance is discussed. The tracing of pulsations in the pulmonary parenchyma is comparable to a plethysmogram and is influenced by both arterial and venous changes in the blood content of the lung. From the tracings the velocity and duration of pulmonary arterial waves have been measured; the speed of the pulse in the lesser circulation has been found to be roughly one-third of the speed of pulse waves in the greater circuit. This is due to a lower pulmonary pressure and greater extensibility of the pulmonary vessels. [Those interested in fluorocardiography should become acquainted with previous work by the same authors on the subject and the appended references will be useful.] *A. I. Suchett-Kaye*

Kymographic Studies of the Function of the Auricle. B. Fabricius. *Acta radiol., Stockh.*, **29**, 152–158, Feb. 28, 1948.

By studying the movements along the left border of the heart between the pulmonary conus and left ventricle with simultaneous kymograms and electro-cardiograms the author has concluded that the kymographic waves at the

level of the left auricular appendix have little significance. Previous workers have described systolic contraction waves arising in the appendix itself and impulses transmitted from the left ventricle and from the pulmonary artery. But owing to the superimposed left hilar shadow and to the small undulations of the auricular appendix the interpretation of these waves has always been difficult. It is probable that the auricular appendix has no independent regular muscular function and therefore no important influence on the circulation. It appears to serve as a complementary space during ventricular systole to preserve the smooth contour of the heart within the pericardial sac. *J. L. Lovibond*

Portacaval Shunts in the Treatment of Portal Hypertension. With Special Reference to Patients Previously Operated Upon. R. R. LINTON. *New Engl. J. Med.*, **238**, 723–727, May 20, 1948.

In the 4 cases reported, portal hypertension was manifested by repeated severe hæmorrhages, for which a variety of previous operations (splenectomy and direct attacks on the varices or their efferent venous trunks) had proved unavailing. A portacaval shunt was then carried out with complete relief in 3 of the patients for the duration of the follow-up periods (14, 14, and 22 months). The most satisfactory portacaval shunt is one in which, after splenectomy, the stump of the splenic vein is anastomosed to the side of the left renal vein. The left kidney should be preserved, since the operation is a severe one and may be followed by oliguria and a rise in the blood non-protein nitrogen even in the presence of both kidneys. Anastomosis of the portal vein to the inferior vena cava may be impossible because of cavernomatous transformation of the portal vein. This operation is also fraught with risk to the bile ducts. The veins of the portal system are devoid of valves, and therefore a shunt downstream from the main trunk may decompress the whole system.

The author emphasizes that previous splenectomy may result in obliteration of the stump of the splenic vein and preclude the establishment of a spleno-renal anastomosis. Therefore the surgeon undertaking a splenectomy for portal hypertension should be prepared to proceed at once to effect a spleno-renal shunt. *Francis F. Rundle*

Dissecting Aneurysms. A Presentation of Ten Case Reports and a Correlation of Clinical and Pathological Findings. A. S. WARREN and A. L. MCQUOWN. *Amer. J. med. Sci.*, **215**, 209–219, Feb., 1948.

The authors describe briefly the pathology of dissecting aneurysm, and discuss the relative importance of atherosclerosis, syphilis, and idiopathic cystic medial necrosis as ætiological factors. They stress the extremely varied symptomatology, and suggest that the clinical manifestations may conveniently be divided into 8 groups, according to whether the symptoms and signs are produced by disturbance of the circulation to the heart, lungs, brain, arm, spinal cord, gastro-intestinal tract, genito-urinary tract, or leg respectively. Histories and necropsy findings in 10 cases are given. The correct diagnosis was made during life in 3 cases, 1 of them at laparotomy. The incidence of dissecting aneurysm during the past 10 years at the authors' hospital (The Charity Hospital of Louisiana) was 1 in 454 necropsies. All the cases recorded in this paper were in males. *A. R. Kelsall*

The Reliability of Percussion of the Left Heart Border. H. E. LEVIN and H. NAGEL. *Bull. Sch. Med. Maryland*, **32**, 221–223, April, 1948.

The authors carried out percussion of the left cardiac border in 25 subjects and compared the position of the outermost point of dullness with the position of the outermost point of the left cardiac silhouette as seen in radiographs taken at 6 feet. They conclude that the heart size was overestimated in about as many cases as it was underestimated, and that the margin of error was not more than 1 cm. in 56% of cases, and not more than 1·5 cm. in 80%. They consider that cardiac percussion of the left border should not be discarded as unreliable. [Two criticisms of this paper are: first, the patients were lean male subjects, and secondly, the majority of these had hearts within normal limits of size. It would have been more convincing if the authors could have shown the value of percussion in cases where the apex beat is not palpable.] *S. Oram*

Effect of Potassium on the Ventricular Deflections of the Electrocardiogram in Hypertensive Cardiovascular Disease. J. M. BRYANT. *Proc. Soc. exp. Biol., N.Y.*, **67**, 557–558, April, 1948.

The author investigated the effect of potassium salts on the electrocardiogram in patients with hypertensive disease and in normal subjects, in doses varying between 10 and 24 g. daily. In the hypertensive group there was a reduction in voltage of the QRS deflections, frequently reduction in length, and shift to the right of the mean axis. An originally inverted T wave became less inverted or upright; it became taller when it was originally upright. In a few instances the T wave became more deeply inverted, though the voltage of the QRS deflection was reduced. All these changes persisted several hours after the administration of the potassium salts. The changes in normal subjects were the same as those in the hypertensive group, but much less pronounced. It is interesting to note that the same type of changes in hypertensive patients can be brought about by a diet with sodium restriction or by sympathectomy. *A. I. Suchett-Kaye*

Cardiovascular System as the Effector Organ in Psychosomatic Phenomena. G. BURCH and T. RAY. *J. Amer. med. Ass.*, **136**, 1011–1017, April, 17, 1948.

The disturbances which emotional stress may produce in the function of the gastro-intestinal and genito-urinary tracts and respiratory and cardiovascular systems is reviewed.

The cardiovascular system is particularly susceptible to psychic disturbances. The effects may be central or peripheral. The central effects include: (1) increased cardiac output; (2) disturbances of cardiac mechanism;

(3) electrocardiographic changes; (4) angina pectoris; and (5) even sudden death. These effects are illustrated by 2 cases in which emotional disturbances produced pain like angina pectoris with transitory electrocardiographic changes (depression of the S–T segment and inversion of the T wave during the attack). The peripheral effects of psychic disturbances are illustrated by 2 cases. A negro showed a typical Raynaud syndrome under emotional strain, and in a medical student erotic thoughts raised the systolic blood pressure from a normal level to 250 mm. Hg and the diastolic pressure to 140 mm. Hg. This rise in blood pressure was associated with a peripheral vasoconstriction, as shown by plethysmographic studies of the index finger.

The use of the plethysmograph in investigating the reactions of the peripheral blood vessels to emotional states and in studying types of personality is discussed. Tension and lack of relaxation in persons may be identified by the plethysmogram. Such methods of investigating the peripheral circulation are considered to offer an objective and quantitative approach to psychiatric problems. The need for psychiatric evaluation and the use of psychotherapy in the proper management of patients with cardiovascular disorders is emphasized.

F. A. Langley

The Behavior of the Venous Pressure during Various Stages of Chronic Congestive Heart Failure. T. B. GIBBONS. *Amer. Heart J.* **35**, 553–566, April, 1948.

Twenty-three patients were studied in whom a state of congestive failure was either induced or improved. Significant changes of degree of congestive heart failure were encountered 34 times; there were 26 instances of recovery and 8 of deterioration. The changes in body weight were taken as an index of the amount of œdema, and serial determinations of venous pressure were made by a direct method. For the most part changes in body weight and in venous pressure ran parallel, but in a few cases venous pressure altered without a change in body weight, and vice versa. When a patient with ascites was improving, the change in body weight might precede the fall in venous pressure. In such cases the venous pressure in the legs was originally higher than that in the arms, but as the ascites disappeared the pressures approached each other. The author concludes that in " all phases of the syndrome of congestive failure changes in the venous pressure and in the degree of congestion are concordant. Any initial discordance in the time relation change in the venous pressure to change in weight associated with œdema can be explained by the state of those tissues affected by œdema."

H. E. Holling

Clinical Electrocardiography. Third Edition. By DAVID SCHERF, and LINN J. BOYD.

It is due perhaps not so much to recent advances in electrocardiography that, since 1940, a third edition has already appeared, as to the increasing demand for a reliable descriptive account of its principles and practice. The new enlarged edition has called for much revision of the former text; nevertheless its authors continue to maintain the critical balance between the theory, physiology and practical application of their subject which remains a feature of their book. The importance of chest leads now meets fuller recognition. More space and greater detail is given to their discussion, particularly in regard to their diagnostic value in myocardial infarction, ventricular hypertrophy and bundle branch lesions. On the other hand unipolar leads are dealt with rather summarily, which is to be regretted in view of their increasing clinical significance. The reproduction of electrocardiograms is technically unpleasing; the majority suffer from over reduction, their definition is indistinct and their full description is often only to be found in the text on another page and not in the abbreviated captions beneath the illustrations. Sometimes the records are presented vertically below each other, but sometimes across the page which is confusing because the leads are not numbered. An adequate bibliography is included at the end of each chapter but unfortunately many references in the text are ambiguous through the omission of dates after authors' names. Attention to these details in the next edition will greatly enhance its merit as a book of easy reference. The text is amply cross-referenced with few misprints and good use is made of emphasis by italics. Some of the chapters, particularly those on extrasystoles, fibrillation and flutter, and the tachycardias are notably lucid.

J. L. Lovibond

Cardiovascular Diseases. Second Edition. By DAVID SCHERF, and LINN J. BOYD.

The original edition of this work consisted of a series of clinical essays on selected cardiac problems written largely from the practical view point in relation to diagnosis and treatment. It has now been rewritten and becomes a complete and eminently readable textbook, retaining many of its former virtues. In the ætiology of certain cardiac symptoms the authors continue to emphasize the importance of the inflammatory myocarditis that occurs after rheumatic fever, tonsillitis, and many other infections, distinguishing it from the primarily degenerative myocarditis of diphtheria and typhoid. One observes that they discard the term " coronary insufficiency " as a false ætiological nomenclature and that they reserve " angina pectoris " to describe a symptom and not a disease entity. The section on Cor Pulmonale could have been accorded more space and the explanations of the " Third Heart Sound " and " Gallop Rhythm " are not too clear. There is a full bibliography after each chapter, but references to authors in the text are mostly made by numbers and not by names, a method now obsolete in this country and one that renders the bibliography less selective. Some unaccustomed terms are used such as " adnexitis," " hypocapnia " (for tachypnœa), and " pathophysiology " though their meaning is usually clear enough. Description of radiology of the heart is limited and the plates that appear are not representative, notably the poor quality illustrations of cardiac aneurysm and atrial septal defect. It would be an advantage if the figures of the cardiographic strips were numbered plainly by leads, and if in the index which is otherwise complete, the main references were emphasized in heavy type. This book should be read in conjunction with the work on Clinical Electrocardiography by the same authors.

J. L. Lovibond

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